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THIS ISSUE

Health Diagnosis in Adults

—A Demonstration in Life Insurance Practice—

The Pneumonokonioses (Silicosis)

Bibliography *and* Laws

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--- from 1556 to December 31, 1933 ---

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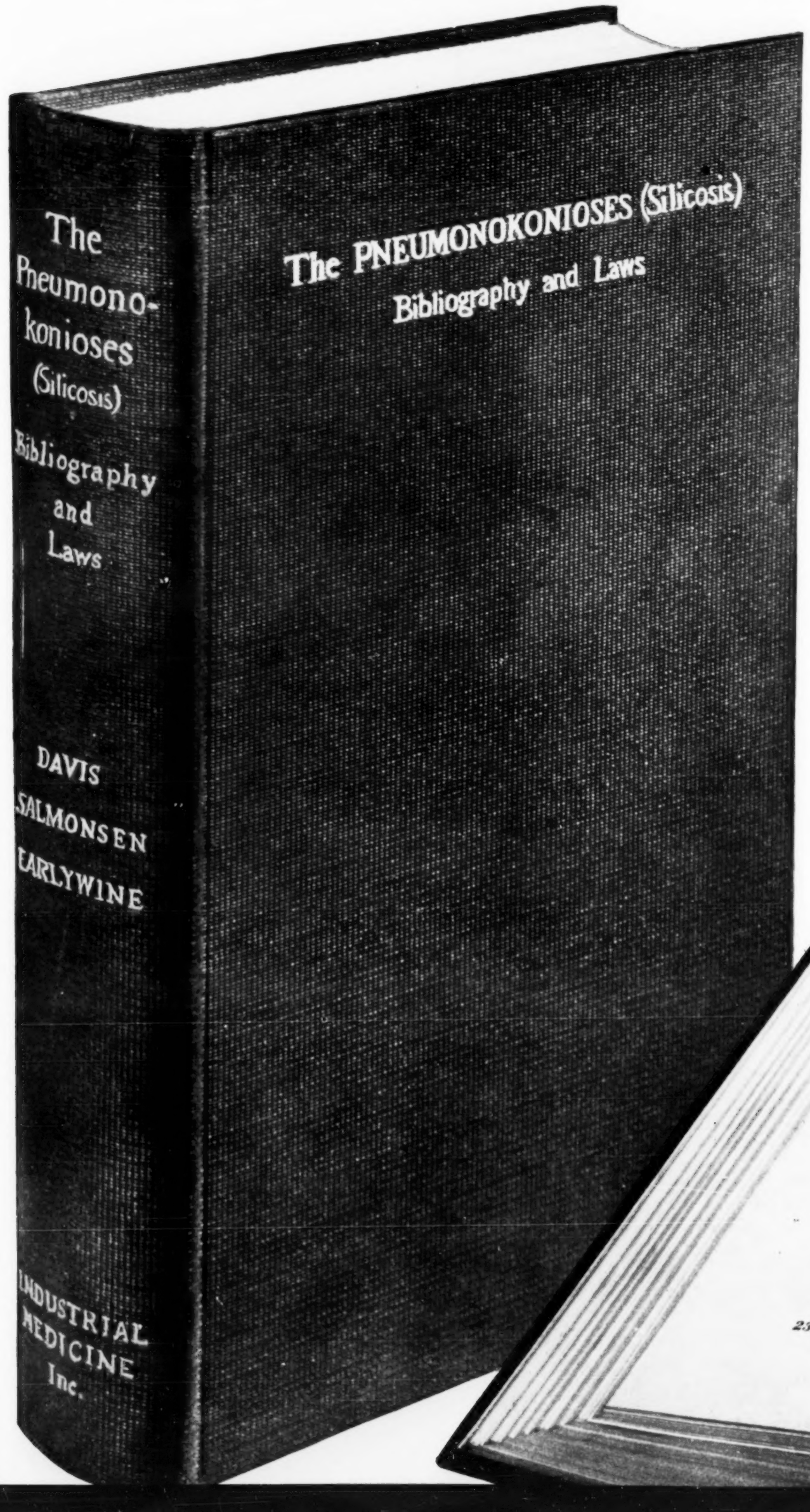

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Industrial Medicine

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"Wherefore to these the Fates shall bend
(And all old idle things—)
Wherefore on these shall Power attend
Beyond the grip of kings:
Each in his place, by right, not grace,
Shall rule his heritage—
The men who simply do the work
For which they draw the wage.
Not such as scorn the loitering street,
Or waste to earn its praise,

Industrial Medicine

Reg. U. S. Pat. Off.

Their noontide's unreturning heat
About their morning ways;
But such as dower each mortgaged hour
Alike with clean courage—
Even the men who do the work
For which they draw the wage—
Men, like to Gods, that do the work
For which they draw the wage—
Begin—continue—close that work
For which they draw the wage!"
—KIPLING

The Journal of Traumatic Surgery and Occupational Diseases

The Law, the Science and the Economics of Industrial Health

Volume 4 NOVEMBER, 1935 Number 11

Health Diagnosis in Adults

*Second Paper**

—A Demonstration in Life Insurance Practice—

by

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Boston,
and
DONALD B. CRAGIN, M.D.,
Hartford

IN JANUARY of this year, because of the results obtained by our physical fitness program among the executives and employees at the Home Office, the Aetna Life Insurance Company decided to offer this program to the larger policyholders in and about Hartford. Accordingly, 350 letters explaining the Physical Fitness Service were sent out as follows:

Office of the President

Dear Mr. .....

For nearly three years the Aetna Life Insurance Company has been giving to its executives and employees a unique service to assist them in advancing and maintaining their physical fitness.

Thus far, over 80% of our executives and 67% of the male employees at the Home Office, beside a goodly percentage of the female employees, have availed themselves of this voluntary service; with such marked improvement in health and efficiency that the company is now offering this service, without charge, to a limited number of its policyholders in the City of Hartford.

This plan is purely an experiment on the part of the company. We think that basically it is sound, and that in offering it to our policyholders we are rendering them a real service. On the other hand, we do not know whether or not it will be acceptable to them. Therefore, we are inaugurating it in Hartford where we are best known and where we feel sure our suggestion will receive courteous and sympathic consideration. Should the plan be acceptable to this group, we will extend it to other localities, but our position will be almost entirely dependent on the manner in which it is received by you.

Frankly, there are two hard-headed business reasons which prompt the company to make this offer:

1. We want you to live longer. Your longer life will appeal most certainly to you, and it will help our business.
2. We consider your good will a valuable asset. If your health is improved by means of this service, we know we will receive your good will.

We are of the opinion from results obtained in our three-year demonstration that from one to 10 years of physical fitness and efficiency can be added to the lives of many policyholders by means of simple changes in habits of living, and that they will make these changes if they are convinced of the need for so doing. This service is suitable for all ages, but is not for treatment of disease. If in our physical check we find you have some physical defect uncorrected, you will be advised to consult your own medical attendant. Contrary to general belief the expectancy of life for the higher ages has not improved a single year in the last half century — in fact, actuarial figures show that the death rate of adults for the decade 1920-1930 has been increasing.

"What is this physical fitness service?" you ask. Briefly, it has to do with finding the answer for you to the following questions:

1. Is your weight suited to your height and build?
2. Have you correctable physical defects that impair your health?
3. Is your daily program of activities faulty?
4. Are your food habits and diet correct?
5. What are your habits as to outdoor living, exercise, etc.?

Those who have availed themselves of this service at our Home Office have brought gratifying results. Among the group there has been a reduction of:

52% in days off from sickness.
199 overweight men have reduced their weight 1,079 pounds.
259 underweight men have gained 802 pounds.

Improvement in physical and mental fitness has been so marked that the voluntary follow-up of the employees to assure themselves of their continuance in improvement average 6.7 return visits.

If he has health, probably a man's best work is done between the ages of 60 and 70 years. From a practical point of view we should not allow ourselves to grow old prematurely, either physically or mentally. In our complicated living today we must fight for health.

The Aetna Life Insurance Company is desirous of helping you.

Whatever your condition of health may be—that of a good athlete or, on the other hand, health such as you do not desire—this physical fitness service will help you. The service is of such a personal nature that the company will consider the records private and accessible only to those in charge of the Department and yourself.

If you wish to avail yourself of this service, please telephone the Department and a time convenient for an appointment will be arranged.

Very truly yours,

.....
President.

THE object of this was to find out first, how many would avail themselves of the service; second, how many would be interested in correct-

* The first paper printed in the January, 1935, number of *Industrial Medicine* reported results obtained with 72 executives and 540 male employees at the home office of the Aetna Life Insurance Company. This work was inaugurated by the Aetna Company solely on the basis of efficiency.

ing physical defects and reorganizing their daily programs in order to correct faulty habits of living.

Fifteen percent of the policyholders thus notified responded.

The following is a report on the first 43 cases. The time covered is from January 29, when the first applicant was examined, to July 12. The group consisted for the greater part of prominent executives and professional men in the community who were carrying from $5,000 to $190,000 of insurance.

General Health Rating

THE health of this group, according to their own ratings, by the scale of excellent, fair plus, fair minus, poor, was as follows:

Excellent	27 men	(63%)
Fair plus	10 men	(23%)
Fair	6 men	(14%)

No one considered his health as below average, or poor.

Their ages ranged from 25 to 63 years—the average age was 42 years.

The physical defects found were as follows:

Hypertension	3
Nasopharyngeal obstruction	2
Dead teeth	6
No lower molars	1
Cerumen	3
Hernia	1
Enlarged prostate	1
Eczema	1
Total	18

21 cases (50%) were seriously overweight.

10 cases (25%) were underweight.

19% of all cases were advised to see their own physicians.

28% of all cases were advised to see their own dentists.

The faulty health habits found were as follows:

	No.	Percent
No regular rest periods	43	100
Insufficient exercise or outdoor sunlight	19	44
Fast eating or washing food down	16	37
Habitual overeating or undereating	10	23
Inadequate vacations or weekly rest	7	16
Excessive tea, coffee, or tobacco	6	14
Eating when overtired	5	12
Irregular bedtime	4	9
Overdoing at work or play	4	9
Candy or sweets between meals	4	9
Working in poor air above 68°	3	7
Worry and fretfulness	2	5
Finicky about food	2	5
Irregular time of bowel movement	2	5
Habits injurious to health	1	2
Irregular habits of living	1	2
Uncontrolled likes and dislikes	1	2
Irregular mealtimes	1	2
Removable physical defects uncorrected	0	0
Sleeping with windows closed	0	0
Total Faulty Health Habits	131	
Average per Person	3	

The chief faulty health habits were those of taking no regular rest periods (100%); insufficient exercise and outdoor sunlight (44%); fast eating (37%); and overeating (23%).

It will be seen from the foregoing that the number of physical defects found was 18, while the number of faulty health habits was 131. Of the 18 physical defects the three cases of hernia, prostate, and eczema were already known to the applicants and were under treatment. The three cases of cerumen were of little significance. From the point of view of disease, nasopharyngeal obstruction and teeth conditions commonly receive little attention in the periodic examination and are frequently overlooked, while from the point of view of impaired health, they are of serious importance. The three cases of hypertension were not the result of disease but of faulty health habits.

Thus it is evident that the results of the usual periodic examination are of little significance as compared with the results of the physical fitness examination which is the basis of health diagnosis. The physical fitness examination includes not only a physical examination for physical defects that cause disease, but also such physical defects as cause impaired health. It includes also a study of daily programs of living to discover such faulty health habits as are direct causes of both disease and impaired health.

Number of Visits

THE total number of return visits (including the first examination) thus far has been 208. Inasmuch as some of the policyholders did not receive notices until April, the eventual number of return visits will be much greater as we are having from five to 10 visits each week from those who have not yet obtained the reduction in weight, or other objects, for which they are working.

Results Obtained

THE method of our Physical Fitness Program is to identify physical defects by a careful physical examination and to discover, by checking up on daily programs of activities, just what habits are essentially faulty. The significance of defects and faulty health habits is then explained to the individual, and definite directions are given for their correction. We find approximately four out of five persons are interested to work for better health provided each can report once a week and is convinced of the efficiency of his new program of living. These policyholders proved to be no exception although the majority of them were busy men with full programs.

A return visit is usually more important than the first examination as it registers a definite effort on the part of the applicant for better health.

The time covered is too short to report end-results, but the following is a statement of what already has been accomplished:

Overweight

TWENTY-ONE applicants were from five to 20 pounds overweight, a total of 181 pounds, an average of 8.6 pounds per person. Of these, 10 reduced their weight 73 pounds, an average of 7.3 pounds each.

Their chief faulty health habits were those of overeating, fast eating, and lack of exercise.

High Blood Pressure

THREE of the overweight cases had hypertension ranging from 160 to 174 mm. One had a diastolic pressure also, of 10 mm.

These men were 33 pounds overweight, an average of 11 pounds per man. They reduced their weight 26 pounds, an average of 8.7 pounds. As a result of their reduction in weight and the correction of their faulty health habits their systolic

pressures dropped 86 points, to normal; an average of 29 mm. per person. Their diastolic pressures were reduced 24 points, an average of 11 mm. per man.

These men have made a total of 35 visits to our department, although two are in business outside of Hartford. One man has not missed a single week since his first examination. The three are carrying a total of $100,000 insurance with the Aetna Company.

According to the sub-standard ratings of the Aetna Company all of these cases were absolute rejects. Aside from their histories, according to the sub-standard ratings of the company, they are all at the present time standard risks.

When an applicant has corrected both his physical defects and faulty health habits, and has attained a normal standard of physical condition, we mark him O.K. Thus far 11 of the 43 men have received this rating.

Amount of Insurance

THESE 43 policyholders were carrying insurance amounting to $1,052,925, an average of $24,580 per person. The saving to the Aetna Company by prolongation of their lives, which can be reasonably expected as a result of correction of physical defects and faulty health habits, is, according to actuarial figures of the Aetna Company, as follows:

Life expectancy prolonged 1 year	$ 78,975
Life expectancy prolonged 2 years	157,950
Life expectancy prolonged 5 years	394,875
Life expectancy prolonged 6 years	473,700

As evidence that life can be prolonged, attention may be called to the 26 hypertension cases among executives and employees, previously reported, that according to estimates from sub-standard figures, show a prolongation of life expectance of more than six years.

Applying this same method of life expectancy to the three cases of hypertension and overweight, found in this group of policyholders, their life expectancy, because of their improved conditions, is prolonged 10 years plus an additional time covered by an increase of $5 per thousand per year added to the cost of insurance. As they were carrying a total insurance of $100,000 this amounts in these three cases alone to $80,000.

These results show that the cost to the company of this Physical Fitness Service is covered by from 1% to 2% of the estimated saving in premiums and interest, as indicated by the actuarial figures of the company. On the other hand, an appreciation of its general value to the policyholders, in improved health, efficiency and happiness, is indicated by the number of their return visits to the department, namely, 208 during the first six months, an average of 4.8 visits per person.

Summary

A PHYSICAL Fitness Service was offered a group of policyholders of the Aetna Life Insurance Company. Fifteen percent of those notified responded.

The results obtained by the first 43 applicants was similar to those obtained by the executives and employees of the company.

Their appreciation of the value of the service was shown by the fact that although these policyholders were busy professional and business men they took time to come to the Aetna Building, which is situated outside the business district of the city, making a total of 208 visits during the first six months of the service, and are continuing to come nine months later.

The 43 policyholders had 18 physical defects and 131 faulty health habits. Twenty-one applicants were from five to 20 pounds overweight, a total of 181 pounds, and an average of 8.6 pounds per person. Ten of these men reduced their weight 73 pounds, an average of 7.3 pounds each.

Their chief faulty health habits were those of overeating, fast eating, and lack of exercise.

Three of the overweight men had hypertension, ranging from 160 to 174 mm. One had a diastolic pressure also, of 100 mm. They reduced their weight 26 pounds, an average of 8.7 pounds, and corrected their faulty health habits, dropping their systolic pressures 86 points, to normal, an average drop of 29 mm. per person. Their diastolic pressures were reduced 24 points, an average of 11 mm. per man. At the end of the six months 11 of the 43 men had so far improved their health by correcting their defects and their faulty health habits as to acquire normal rating.

According to the actuarial ratings of the company, the policyholders increased their life expectancy sufficiently to indicate a saving to the company in interest and dividends to such an amount that from 1% to 2% of this saving would cover the entire cost of the service rendered.

Comments

ASIDE from the financial value of this service to the company, because of increased life expectance, is the asset of good will and renewed contacts. Both by personal letters to the president of the company and by remarks of practically every policyholder taking the service, appreciation has been expressed as to the value of the service.

By means of this service, life insurance becomes life assurance, a sounder basis for insurance practice.

Life insurance business has increased to its present magnitude because of its great service in safeguarding the home against the vicissitudes of old age and death. If prevention of sickness and death can be added to this service rendered policyholders, the writers believe a constructive, forward step will be taken in insurance practice.

Undulant Fever

OLIN states* that from 1929 to 1933 about 120 cases of undulant fever have occurred annually in Sweden. Of 565 patients, 65.2% were men. Persons in the fourth decade of life were most often affected. A close relation between undulant fever and the infectious abortion in cattle was noted. Serologic studies indicated that latent abortive infection with Bang's bacillus occurs in man. Test of 288 persons without history of the fever and with little likelihood of exposure to infection gave positive cutaneous reactions in 21, or 7.3%, the reaction being more often positive in adults than in children, and in women than in men. The cutaneous reaction was positive in six persons with a history of undulant fever from six months to six years previously and in nine bacteriologists who had been

* *Svenska Lakaresallskapets Handlingar,* Stockholm; Abstr. *J.A.M.A.* (August 31, 1935).

working with Brucella cultures for a longer time without having undulant fever. A marked phagocytosis was found in four of the six and in seven of the bacteriologists. Of 251 attempts at blood culture in undulant fever, 46.2% were positive. Bacteria were more often demonstrable in a highly febrile stage but could occasionally be cultivated from the blood during afebrile periods. In 75 cases verified by blood cultures, the results show that agglutination in serum dilution is 1:320 and from 0 to 10% hemolysin in complement fixation tests with 0.1 c.c. of serum could be regarded as positive. The agglutination test seemed somewhat more reliable. In 171 cases of undulant fever there was a single fever period in one-fifth, there were two or three fever periods in more than one-half, and there were four or more fever periods in one-fourth. The fever was usually high. The general condition was little affected. The pulse frequency was low in comparison to the temperature. Enlargement of the spleen was found in more than one-third of the cases. The diazo reaction of the urine was positive in about one-fourth of 64 cases. Leukemia appeared in most cases; the percentage of mononuclear elements was generally increased, and slight or moderately increased blood sedimentation was usual, the values generally being lower during the first stage than during the later course and higher during the febrile than during afebrile periods. In the six fatal cases the cause of death was mainly pulmonary or circulatory complications. The duration of the disease till disappearance of the fever varied from eight to 532 days, the average duration being 91.5 days. The average convalescence lasted three months. Chemotherapy and vaccine treatment with subcutaneous injection of killed bacteria were both without effect.

Irritants in Adhesive Plaster

SKIN reactions following the use of adhesive plaster are of frequent occurrence. Often this manifestation of the skin is not only the cause of great discomfort to the patient but actually interferes with the plan of treatment.*

In making patch tests, reactions from adhesive plaster often occur and are not only annoying to the patient but may interfere with the reading of the action. Shelmire[1], in a recent article, summed up the obstacles that irritation from adhesive plaster presents in the field of patch testing and advocated a substitute for the adhesive plaster. This study was undertaken with the purpose of determining, if possible, the irritating substances in adhesive plaster so that an intelligent effort could be made by manufacturers to eliminate them or provide harmless substitutes.

A number of observers have been interested in the causes of dermatitis produced by adhesive plaster. Bloch reports that 1% of the normal population develops dermatitis from adhesive tape. Siemens[2] tested susceptible cases with the ingredients of adhesive plaster and came to the conclusion that dammar resin was responsible for some of the irritating qualities. He believed that the reaction was not based on idiosyncrasy but was really due to direct irritation. Kilmer[3] stated that, as the result of his investigations, the ingredients of adhesive tape are not irritating as such. He believes that the skin secretions are retained under the moisture-repellent coating, with a resultant maceration of the epidermis. He states that this, rather than idiosyncrasy, is the most frequent cause of the irritation. He also states, however, that there might be a few instances of reactions due to adhesive plaster which are based on specific hypersensitivity.

In our own observations the skin manifestations following applications of adhesive tape can be roughly divided into two types: In one we have erythema and, in some cases, even edema and vesicles which are due to direct traumatic irritation as the result of the application of a firmly adherent substance to the skin with resultant trauma on its removal. This reaction is usually fleeting in character or, at the most, subsides after two or three days.

The other type of reaction due to adhesive is caused by hypersensitivity to one or more of the ingredients of the plaster and is a dermatitis venenata, or a contact eczema. This type of reaction usually increases in severity after the removal of the plaster and lasts for a considerable period of time. In many cases the severity of the reaction increases with the continued use of the adhesive plaster.

THE methods for the manufacture of adhesive plaster are more or less secret. No textbooks could be found describing the process. A number of firms manufacturing adhesive plaster in the United States were informed as to the purpose of this study and were asked to describe their method of manufacture and to give us a list of the ingredients which they used. A number of them listed and sent samples of the ingredients used, and one manufacturer permitted us to inspect his method of manufacture and to do the patch tests required on volunteer workmen from the factory. The ingredients used are listed below. All of these were not used by any one manufacturer.

1. Rubber:
   a. South American Para rubber.
   b. Plantation smoked sheet.
   c. Balata rubber.
   d. Gutta siac.
2. Rosin, grade I.
3. "Burgundy" pitch.
4. Olibanum.
5. Beeswax.
6. Zinc oxide.
7. Anhydrous lanolin.
8. Starch.
9. Orris root.

A homogeneous mass is made by milling rubber, butta siac, or balata, with adhesives such as rosin, pitch, and olibanum, fillers such as orris root, starch, and zinc oxide. Beeswax and lanolin are also added for other purposes. This homogeneous mass is spread by calender machines on suitable fabrics.

FOR the purpose of this study 120 employees of a plant manufacturing adhesive tape were used in the experiment. Eight varieties of adhesive plaster manufactured by six different companies were obtained and placed as patches about one inch square on the arms and backs of these test subjects. They were left on for 48 hours, at the end of which time 50 of the patients showed a reaction to one or more of the adhesives applied.

There was no marked difference in reaction to any particular adhesive. The least number of re-

* LOUIS SCHWARTZ, senior surgeon, United States Public Health Service, and SAMUEL M. PECK, Assistant Clinical Professor of Dermatology and Syphilology, New York University; Pub. Health Reports, Vol. 50, No. 24, June 14, 1935.

actions obtained from any adhesive was 16%, and the greatest number from any one adhesive was 20%.

The sites of the patches were again inspected two days after the removal of the adhesive. At that time 13 of the 70 patients in whom no reaction had been observed at the end of the 48-hour period showed late reactions. Some of the reactions seen upon the removal of the plaster had become intensified. It was interesting to observe that, in a number of instances where only a few of the adhesives seemed to give a reaction there was a delayed reaction to all of the previously inactive adhesives.

The reactions observed varied from a slight erythema to an erythema with edema, papules, and vesicle formation.

For the purposes of this study, the patients were divided into three classes:

Class A.—In this group were placed those who showed marked reactions at the first removal of the adhesive tape with continued intensification at the second inspection.

Class B.—In this group were placed those who showed a negative or only a slight erythema at the first inspection but who later developed delayed reactions.

Class C.—Patients who at no time showed anything more than varying degrees of erythema at the site of the adhesive patch.

Twenty-one of the 63 patients who had showed reactions volunteered for further patch testing with the ingredients of the adhesive plasters. Six of these were in Class A, 12 in Class B, and three in Class C.

It was not possible to test all of these cases, especially the women, with more than five of the 11 ingredients which we wished to study. However, in each instance where only a limited number of tests could be made, those substances were tried which we thought were responsible for the irritation. One of the 10 men tested had 12 patches placed on his back, because he stated that he was sensitive to raw South American Para rubber biscuits, and a piece of this material was used on him as a patch test.

Substances used in patch tests:

1. South American Para rubber, which had been milled, washed, and dried, ready to be incorporated into the adhesive mass.
2. Starch.
3. Lanolin.
4. Orris root.
5. I-Rosin.
6. Olibanum.
7. Gutta siac.
8. Beeswax.
9. Burgundy pitch.
10. Zinc oxide.
11. Wood rosin extracted from stumps of pine trees.

The patches were left on for 48 hours and the reactions read. They were inspected for late reactions 72 hours after the patches had been removed.

The rosins used in the manufacture of adhesive plaster belong to the class of natural resins. These rosins are divided, according to T. Hedley Barry[4] into eight classes, with relation to their hardness, No. 1 being the softest:

1. Dammar resin;
2. Shellac;
3. Mastic;
4. Sandarac;
5. Rosin;
6. Elemi;
7. Turpentine oleo resin;
8. Burgundy pitch.

Rosin is obtained from trees of the order of Coniferae, genus *Pinus*. All pines may be used, but most of the rosin in the United States is collected from the long leaf and the short leaf pines. The trees are scarred, and the exuding gum is collected, and purified by filtration, sedimentation, and distillation, removing the turpentine which is the principal product. The residue, called colophony, is the source of the different grades of rosin. The rosin collected the first year that the tree is tapped is light in color and is graded by the manufacturers according to color from WW to K. The second season that the tree is tapped, the rosin obtained is darker and more viscous and is graded by the manufacturer from I to G. With successive tappings, the sap obtained contains less turpentine and less rosin.

Rosins contain a number of oils and acids. The principal ones are kidney oil, bloom oil, abietic acid (alpha, beta, and gamma), pinnic acid (alpha, beta, and gamma), sylvic acid, and abietic anhydride.

Wood rosin is a name applied to rosin extracted by a special process from the stumps of pine trees. It is very similar to ordinary rosin.

Olibanum is a gum resin obtained from the exuded juice of a tree belonging to the genus *Boswellia*, which grows in East Africa and the southern coast of Arabia. It is pale yellow, has a pleasant aromatic odor, and is used only in certain varieties of plaster so as to give them a pleasant odor.

South American Para rubber, which comes to the United States in so-called "biscuits," is obtained by tapping the rubber tree and is cured over a small fire made of the fruits or nuts of the urucuri. This fire gives a dense smoke rich in the products of distillation, such as creosote, tarry matter, and acetic acid. A long wooden rod, or mandrel, with a paddle attached, is covered with a thin film of the latex collected from the tree. This is rotated in the smoke until the latex sets, when a fresh layer of latex is poured over the first and the process repeated until a biscuit of smoked Para rubber, weighing from 20 to 100 pounds, is built up. Such a biscuit of rubber, by the very nature of the curing method, is saturated and impregnated with creosote, tarry matter, and acetic acid.

Plantation rubber is obtained from the Malay Peninsulas, the East Indies, and Sumatra. The latex is collected and is coagulated by the addition of dilute acetic acid. After the coagulum is formed, it is removed from the serum and passed through washing roller mills, which squeeze out the mother liquor and wash out extraneous materials. The sheets are then hung up to dry and are frequently smoked during the drying period by burning coconut husks and hard wood. The products of this smoking are only on the surface of the crepe formed sheet and are not impregnated into the rubber itself, as is the case with the South American Para rubber. Plantation rubber is dry and clean, while the South American Para rubber contains moisture, sand, stones, bark, and other impurities which must be cleaned out before it is used. While plantation rubber contains about 6% of impurities, South American Para may contain anywhere from 12 to 40%.

Studies made in tire-manufacturing plants, where crepe and smoked sheet rubber are exclusively used, fail to show any dermatitis among those handling the raw rubber, whereas in the course of the present studies we found one worker

who develops a severe dermatitis very time he handles South American Para rubber biscuits.

Balata is the product obtained by coagulating the latex of *Mimusops globosa*, a large forest tree belonging to the order of Sapotaceae, a native of British, Dutch, and French Guiana, and Trinidad, Jamaica, and Brazil. It resembles true gutta percha in physical properties, and the tree yielding it belongs to the same order which furnishes gutta percha (*Palaquium* spp.). Balata, like gutta percha, consists of a hydrocarbon $C_{10}H_{16}$, associated with resins, but contains a higher percentage of resins than gutta percha. The resins in balata are similar to those in gutta percha and consist of—

(1) Albane, which is soluble in hot alcohol.

(2) Fluavile, which is soluble in cold alcohol.

Gutta siac is very similar in its properties to balata and gutta percha.

The so-called "Burgundy" pitch used in adhesive plaster manufacture does not necessarily come from Burgundy. That which we tested was a mixture of resins and other substances, the composition of which is kept secret by the makers.

Beeswax, zinc oxide, lanolin, starch, and orris root need no description.

CLASS A.—The six persons patched in this class were males and were patched with all of the 11 substances listed above. (Altogether there were seven cases in this group, one of them a woman who would not submit to more than the original tests with the eight varieties of adhesive.) We thought that the reactions in this group were due to hypersensitivity. In all of these cases we had erythema, endema, papules, and vesicles which did not disappear but went on to eczematization. As can be seen from Table 1, there was not a single instance in which there was sensitization to less than two of the ingredients used as patches. All six were sensitive to "Burgundy" pitch. Three showed marked positive reactions and two showed questionable reactions to South American Para rubber that had been milled, washed, and dried. Three showed positive patch tests to wood rosin obtained from pine-tree stumps. Two gave positive reactions to olibanum, two to beeswax, and one each to lanolin, orris root, I-rosin, and gutta siac.

Class B.—Altogether there were 34 cases put in this class. Of this number, 12 consented to further patch testing—11 women and one man. The man was patched with all 11 of the ingredients and the women with only five of them, namely, (1) South American Para rubber, which had been milled, washed, and dried; (2) I-rosin; (3) "Burgundy" pitch; (4) zinc oxide; and (5) wood rosin.

In this class we thought that we were dealing with reactions of hypersensitivity of the delayed type, because the delayed reactions in these cases were more pronounced than were the ractions seen immediatly upon removal of the patches. These delayed reactions also showed erythema, edema, papules, and vesicles which persisted for a number of days. These may be the types of cases which become more and more sensitive to adhesive tape, depending on the duration of the application and the number of times within a given period that the adhesive is applied.

One of the women tested showed such a generalized reaction that it was impossible to differentiate between the individual patches. Six of the women showed positive reactions to one or more of the patches. Two of them showed questionable reactions to one of the patches, and two showed no reactions to any of the patches. The fact that no reactions, or only questionable ones, resulted in four of these women, may be interpreted either as a possible sensitization to one of the ingredients of adhesive plaster with which they were not patched or to the fact that their reactions in the first series of tests were due to summation of effects from several of the ingredients in adhesive plaster. In this group, when the patches were first removed there were two reactions to rubber, four to I-rosin, two to "Burgundy" pitch, and two to wood rosin. The sites were again inspected 72 hours after the removal of the patches. At this time the original reactions were still present and eczematoid in character. In addition there were three subjects who showed a questionable reaction to rubber, one individual who showed a positive reations to I-rosin, and three more showed reactions to wood rosin. These, of course, were delayed reactions.

Class and Subject	S. A. Para rubber	Starch	Anhydrous lanolin	Orris root	I-Rosin	Olibanum	Gutta siac	Beeswax	"Burgundy" pitch	Zinc oxide	"Wood" rosin
	Substances for patching										
Class A											
J.B.C. (M)	?	—	—	—	—	—	—	—	+	—	+
E.J.D. (M)	—	—	—	—	—	—	?	+	+	—	+
W.F. (M)	+	?	+	?	—	+	—	—	+	?	—
D.D. (M)	+	—	—	—	—	—	+	—	+	—	—
J.F.B. (M)	?	—	—	—	+	+	—	+	+	—	—
M.A. (M)	+		?	+	?			?	+		+
Class B											
H.A. (M)	—	—	—	—	+	—	—	—	—	—	—
G.K. (F)	—	0	0	0	+	0	0	0	—	—	—
C.D. (F)	+	0	0	0	+	0	0	0	+	—	+
G.V.S. (F)	?	0	0	0	—	0	0	0	—	—	+
M.C. (F)	?	0	0	0	+	0	0	0	—	—	+
L.O.H. (F)	+	0	0	0	—	0	0	0	—	—	+
H.K. (F)	?	0	0	0	+	0	0	0	+	—	+
C.H. (F)	—	0	0	0	—	0	0	0	—	—	?
M.F. (F)	—	0	0	0	—	0	0	0	—	—	—
E.W. (F)	—	0	0	0	—	0	0	0	—	—	—
G.O.L. (F)	?	0	0	0	—	0	0	0	?	—	—
M.E.L.[1] (F)											
Class C											
O.L.V. (M)	—	—	—	?	—	—	—	—	—	—	?
G.R. (M)	—	—	—	—	—	—	—	?	—	—	—
F.B. (M)	—	—	—	—	—	—	—	—	?	—	—
Total	5	1	1	6	2	1	2	8			8
Percent	25	5	5	30	10	5	10	40			40

[1] Reaction too general to be read for individual patches.
+ = Positive reaction.
— = No reaction.
? = Faint erythema; doubtful reaction.
0 = Not patched.

Table 1.—Summary of Reactions

Class C.—We were able to obtain only three men in this group for further patching. They were patched with all of the 11 ingredients listed, and in no instance could we obtain a real positive reaction. One gave a questionable reaction to or-

is root, one a questionable reaction to beeswax, and one a questionable reaction to zinc oxide. We believe that the original reactions in this group to the eight patches of adhesive were due purely to mechanical irritation of the plaster and maceration of the skin. None of these reactions lasted so that they could be seen 72 hours after the patches had been removed.

TWENTY-ONE subjects showing various degress of adhesive plaster reaction were tested with 11 ingredients of adhesive plaster. One of these developed a generalized reaction so that individual tests could not be evaluated. Seven of the remaining 20 were negative to the patch tests. Of the 13 remaining, eight showed positive reactions to wood rosin extracted from the stumps of pine trees; eight to so-called "Burgundy" pitch; six to I-rosin; five to South American para rubber, which had been milled, washed, and dried; two to beeswax; two to olibanum; and one each to lanolin, orris root, and gutta siac.

All of the subjects in class A showed positive reactions to one or more of the rosins, and 50% were sensitive to rubber.

Seven of the subjects tested in class B were sensivtive to one or more of the rozins, and two were sensitive to rubber.

The tests seemed to incidate that there are two types of reactions to adhesive tape: One is purely chemical and due to resultant maceration and mechanical trauma from the application and the removal of the plaster, and the other is due to hypersensitivity to one or more of the ingredients of the plaster. The results indicate that the chief irritants in the adhesive plasters that we tested are the rosins, in which can be included the so-called "Burgundy" pitch, and the smoke-cured wild rubber, of which South American Para is an example.

An attempt was made to determine whether complexion or previous diseases of the skin or an allergic diathesis had a predisposing effect on sensitivity to adhesive plaster. All the subjects patched with adhesive plaster were questioned as to these facts. No such correlation could be established.

CONCLUSIONS: 1. Skin reactions following the use of adhesive plaster are of frequent occurrence.

2. There are two kinds: (*a*) Due to traumatic phenomena and maceration resulting from the application and removal of a firmly adherent material; and (*b*) an eczematoid reaction due to hypersensitivity to one or more of the ingredients of the plaster.

3. The reaction classed under 2 (*a*) disappears shortly after the removal of the plaster.

4. The reaction classed under 2 (*b*) persists for many days.

5. The chief irritants in adhesive plaster have been found to be the resins and the smoke-cured wild rubber.

6. It is obvious that the irritation due to the tackiness of the adhesive cannot be avoided. It seems, however, that research in adhesive manufacture should make it possible to substitute nonirritating types of resins and rubber for the present types used.

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Skin Irritants in Adhesive Plaster

THE occurrence of skin reactions[1] following the use of adhesive plaster is by no means uncommon indeed, the statement has been made that approximately 1% of all normal individuals show the phenomenon. According to the current opinion, two types of reactions may occur.

One type is simply the result of mechanical trauma by the adhesive plaster and it disappears within a short time after the removal of the plaster. The other type of reaction is a true allergic response to substances present in the adhesive base. Such reactions often increase in severity after removal of the plaster and may persist for many days. Obviously, little can be done to decrease the incidence of the reactions due to tissue trauma; however, the occurrence of the allergic manifestations might be lessened if the nature of the irritating substances were known. A recent investigation on this problem has yielded interesting information. The effects of the various constituents of several different makes of adhesive plaster were determined in 21 subjects who had previously been shown to react to the mixture of substances present in the original tape. Eleven ingredients, namely, three kinds of rubber, pine wood rosin, "Burgundy" pitch (a mixture of resins), olibanum (a gum resin), beeswax, zinc oxide, wool fat, starch and orris root, were tested. They were applied to the skin of the arm or the back. A large number of skin reactions resulted from the application of rosin, "Burgundy" pitch, and the smoke-cured wild rubber. Reactions to the other constituents were fewer and less pronounced. Therefore it appears that the substances responsible for the allergic reactions are chiefly the resins and the wild rubber. These observations should stimulate research in the industries for the purpose of finding nonirritating types of resins and rubber to replace those now used in the manufacture of adhesive plasters.

Tested Tape

ONE of the most common medical procedures today[2] is the skin test by means of which doctors tell whether a person is sensitive to ragweed, strawberries, horsehair, chicken feathers, scarlet fever, diphtheria or any other known allergen. The physician scrapes off a tiny area of the patient's skin, applies a drop or two of the allergic substance, covers the whole with a piece of adhe-

1. *J.A.M.A.*, August 24, 1935.
2. *Time*, July 8, 1935.

sive plaster. Skin tests have preserved the health and lives of multitudes. They have also served to reveal that about 1% of the population develops an eczema-like skin irritation solely from the adhesive tape which is used in covering the skin-test material.

The few U. S. concerns that manufacture surgical adhesive tape paid practically no attention to the dermatitis which their products occasionally cause until Surgeon General Hugh Smith Cumming of the U. S. Public Health Service ordered an investigation. Dr. Cumming appointed Dr. Louis Schwartz of his own staff to do the work and Professor Samuel Mortimor Peck, Manhattan dermatologist, to help. They published their report last week.

Drs. Schwartz and Peck first queried the most revered pharmaceutical chemist in the country, Frederick Barnett Kilmer, 83, head of Johnson & Johnson's laboratories at New Brunswick, N. J. since 1889. Mr. Kilmer told them that, as the result of his investigations, he considered the ingredients of adhesive tape not irritating as such; that the skin secretions are retained under the moisture-repellent coating with a resultant maceration of the epidermis. This, rather than idiosyncrasy, said Mr. Kilmer, is the most frequent cause of the irritation.

Drs. Schwartz and Peck found the manufacturers of adhesive tape as secretive about the ingredients and methods of manufacture as they are about the yearly yardage and dollar value of their plaster.

Eventually, however, the following list of ingredients became clear: rubber, rosin, "Burgundy" pitch, olibanum, beeswax, zinc, oxide, anhydrous lanolin, starch, orris root.

Appropriate tests of those ingredients made separately on the skins of volunteers demonstrated that, apart from maceration and mechanical injury, rosins, pitch and smoke-cured wild rubber are the chief irritants. Complexion, previous skin diseases or a prediposition to shingles or other allergens apparently have nothing to do with the sensitivity to adhesive plaster found in one out of every hundred normal individuals, men, women and children.

Pointing a reproving finger at adhesive tape manufacturers, the Schwartz-Peck report to Surgeon General Cumming concludes: "Research in adhesive manufacture should make it possible to substitute non-irritating types of rosins and rubber for the present types."

To the Editor of *Industrial Medicine:*

ARE these comments concerning skin irritants in adhesive plaster of any use to readers of *Industrial Medicine?* They came to me in a personal communication from a Massachusetts colloid chemist, who has had extensive experience in the manufacture of rubber and adhesives, and has paid close attention to dermatoses, after he read *TIME's* report on Schwartz and Peck's recent U.S.P.H.S. report on the subject. My communicant is Morris Omansky, 9 Babcock St., Brookline, Mass.

His comments:

"1. South American Para rubber is not a simple substance. Its irritating action may be due to non-rubber impurities.

"2. There are no 'kidney' or 'bloom' oils in existence in rosin or elsewhere. The use of these words reveals an inexact reading of some chemical text books.

"3. South American Para rubber in spite of its method of preparation is not saturated with creosote, tarry matter and acetic acid. The doctors are jumping at conclusions.

"4. Gutta Siak is very dissimilar in properties to balata and gutta percha in spite of its name and superficial appearance. Hence it cannot be used for cables or golf balls.

"5. No allowance is made for mutual protection of ingredients by each other, a common phenomenon which makes paint practical. The interaction between zinc oxide and acidic substances is overlooked.

"6. Most serious is the ignoring of the myriads of bacteria present in some of the substances. Thus an employee of mine lost his life through infection from gutta Siak (not siac)."

—Myron Weiss, Associate Editor, *TIME.*

Modified Schafer Method

of Artificial Respiration

THE defects of the Schafer method in cases of asphyxia are pointed out in a paper by Prof. Hederer of the French Naval School at Toulon, read at the May 14 meeting of the Academie de medecine.* The method suggested must be carried out by two persons instead of one, which is its only disadvantage over the Schafer method, obligatory in France since 1927. In the latter method the individual to whom artificial respiration is being applied lies face downward with the head turned to one side. By compression of the lower ribs, during expiration, an attempt is made to stimulate the diaphragmatic reflex. The criticism which Hederer makes of the Schafer method is that the volume of air entering and leaving the lungs is only about 100 to 150 cc. as compared to the normal volume of 500 cc. and one of 250 to 280 cc. when the Silvester method is used. Another criticism is that the pulmonary circulation is stimulated much less with the Schafer than with the Silvester method.

These criticisms are based on laboratory studies in which the two methods were compared. Hederer suggests a method of artificial respiration combining, from a physiologic point of view, the advantages of the Silvester method with the preferable posture of the Schafer method. With the asphyxiated or drowned person in the prone position, one assistant exerts pressure on the last ribs with the fingers of both hands, the thumbs resting near the median line of the back. At regular intervals (Schafer method) expiratory efforts are imitated by pressure on the lower part of the thorax. The second assistant, kneeling at the head of the person to be resuscitated, grasps both elbows and pulls the arms upward without changing the position of the head and hands of the subject. Thus inspiratory and expiratory movements are alternately imparted to the thorax by the two assistants. The volume of air rises from 100 to 150 cc. to 250 to 280 cc. and the intracardiac pressure is greatly increased.

It is as easy to teach this method as to teach the Schafer method alone.

* Paris Correspondent, *J.A.M.A.* (August 24, 1935).

Industrial Manganese Poisoning

—With a Review of the Literature—

by

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MANGANESE was discovered by Scheele in 1774. Manganese is not found native but is widely diffused as it accompanies nearly all iron ores. The chief minerals in which it is found are the sulphide in alabandite, (MnS); hauserite (MnS_2); oxides as pyrolusite (MnO_2); braunite (Mn_2O_3, $3Mn_2O_3$, $MnSiO_3$); manganite (MnOOH); hausmannite (Mn_3O_4); franklinite ($FeZnMn\ (FeMn)_2O_4$); psilomelane (MnO_2 (+H_2O, K_2O, or BaO); wad (a mixture of manganese oxides, such as cobalt, copper and lead); as the carbonate in rhodochrosite ($MnCo_3$); as a silicate in tephroite (Mn_2SiO_4), knebelite ($(FeMn)_2SiO_4$), heterocline, helvine, and rhodonite ($MnSiO_3$ or $MnOSiO_2$); as a phosphate in triplite ($(RF)RPO_4$) (R chiefly representing manganese and iron).

The principal use of manganese minerals is in the production of alloys with iron spiegeleisen and ferromanganese, used in the manufacture of steel. It appears to unite with iron in all proportions. It retards the oxidation of iron in operations such as puddling, Bessermerizing, and open hearth methods of making steel. It also appears to volatilize to some extent in these operations,[1] a possible factor in poisoning.

Manganese has a valence of 2, 3, 4, 6, 7 and consequently has a great number of compounds which it may form. Manganous oxide (MnO) represents the only base capable of forming stable salts, manganic oxide (Mn_2O_3), trimanganese tetraoxide (Mn_3O_4), manganese peroxide (MnO_2), manganese trioxide (MnO_3), maganese heptoxide (Mn_2O_7), and manganese teroxide (MnO_4). It forms a large number of salts, of which the manganous salts are derivatives of the trivalent maganese. In the manganates, the derivatives form manganic acid (H_2MnO_4), which is not known in a free state. Other uses are in the manufacture of dry batteries, chlorine, oxygen driers for varnish; as a decolorizer for molten glass, to color glass pottery and bricks; in making paints and enamels, and in dyeing calico and linoleum. A small quantity finds use in the coloring and graining of soaps in the aniline and alizarin factories.

Manganese, like silica, appears to be found in all plant and animal tissue. Maumene[2] found it only sparingly in the potato, while in the sugar beet he found it in considerable amounts. In the ash of the tea leaves Maumene found 10% of manganese. Von Schroder[3] found 33% of manganese dioxide in the ash of a 90-year-old white pine tree, which had grown on brownstone. In milk, urine, bones and hair it is found only in traces. It is a normal constituent of the blood of man and animal. Bertrand[4] in 1905 began a series of exact scientific research on the subject of manganese. These papers deal with the effect of the catalytic action of traces of manganese on the growth of certain molds, plants and bacteria. Bertrand's work laid the foundation for the more recent research upon the physiological role of manganese in the human economy.

THE medical profession first learned of manganese poisoning when Couper, in 1837,[5] reported four cases of chronic poisoning and one case of acute poisoning in a man who had ingested an ounce of manganese with only a purgative effect. Couper reported some observations he had made with manganese peroxide that were of great toxicological interest. A considerable number of men were grinding manganese dioxide for the manufacture of chlorine for bleaching powder. He stated that these workmen were exposed to the dust, and that their skin was constantly covered with a heavy layer of this oxide and that they breathed air contaminated with a multitude of particles. (In spite of the bad working conditions he reported only four cases of intoxication.) The first case to come under his observation was that of a young man in 1821, who had been previously in excellent health, began to work with manganese dioxide and in a few months time showed symptoms of paraplegia, which after a certain time became so severe he was forced to give up his work. He tried many remedies used at that time for paralysis, even leaving his home for a rest for one year, at the end of which time he returned having made but little progress. The following year another workman who was also grinding manganese and who had been previously well, took ill very suddenly. He did not suspect that this substance was toxic and therefore he continued his work, but he received various treatments. As the paralytic process developed, manganese was suspected as being the toxic agent. This workman went to another country but his condition did not improve. He recovered after six years.

Couper describes his observations as follows: The muscular weakness was usually more pronounced in the lower extremities; this weakness was so severe that the patient's legs could not support the body; the victim usually spoke with a lamenting voice and could be understood only with difficulty. There were no sensory or mental disturbances. The face musculature appeared to be paralyzed. He was salivating while speaking. There were no tremors in any other parts of body. He had no colic and he was not constipated and there were no derangements of the digestive functions. For treatment he was given mercurials, warm baths and vesicants which were applied at the head and spinal cord, and also strychnine. None of these remedies gave any result.

In addition to the cases above described he observed two other workmen who were grinding manganese ores; both had similar experiences. But in these cases the process was arrested because the workmen were assigned to another plant as soon as the symptoms developed. They all recovered within a few weeks. Couper also noted that the paralysis caused by manganese differs from that produced by lead in that the former does not act at all on the intestinal tract producing colics as lead does (saturnism). Manganese, furthermore, never produces constipation as lead does.

In 1860, Wichert[6] reported the excretion in bile of soluble manganese salts. Turner, in 1860,[7] reported the elimination of manganese when employed medicinally. He states that it is found in hair, bones, epidermis and also in the gastric juice. He also found it to be one of the components of blood, gall stones and urinary calculi, but did not find it in urine. He found that it always existed in association with iron. He thinks that this metal is eliminated by means of the liver and hence is most detectable in the bile. He mentions the early therapeutic use of manganese in chlorosis used alone or in combination with iron. Schlockow[8] described a peculiar and very slowly developing poisoning in Silesian miners working with ore containing zinc, manganese, lead, arsenic and cadmium. The characteristic complex of these workers occurred after 12 years of mining. They complained of pains in the back and hyperesthesia of the lower extremities. Reflexes were somewhat increased. Muscular weakness, positive Romberg. There were disturbances in gait; the patient had difficulty turning around, and had a peculiar slapping gait. There was no atrophy in the muscles. Finally there were severe parietic symptoms of the extremities. Schlockow considered this to be a spinal cord disease. It would be difficult for one to say absolutely that it was manganese. Many of these same symptoms could be caused by the lead and arsenic present. I think manganese played a very important role in these cases. Richet,[9] in 1881, found that the maximum amount of manganese which a fish would tolerate for 48 hours was 0.30 of a gram of manganese chloride.

The next contribution was made by Kobert,[10] in 1883, who observed that manganese when introduced into the blood stream was very poisonous, but was non-poisonous by the stomach. Also, that this metal as with all other heavy metals, (except iron) causes parenchymatous and interstitial nephritis. Reinaud,[11] in 1883, discussed manganese from a minerological, chemical and physiological point of view. Cahn,[12] in 1884, using rabbits, injected intravenously and subcutaneously a solution of manganous oxide. He summarized his results by stating as follows: (1) If manganese be introduced into the blood stream it is not taken up by the blood cells. (2) The metal is taken up by the parenchymatous organs whence it is transported further; the major part is eliminated through the gastro-intestinal tract and appears in the feces. (3) Manganese is not absorbed through the intact intestinal wall, at least not in significant amounts.

OWING to the chemical relationship of manganese to iron, the author thinks that the same conditions are true also for this latter metal. He found in the serum .0159 grams of manganese oxide in 60 cc. of blood.

Schreiber,[13] in 1901, in association with Harnack, used rabbits which were fed manganese peptonate. After four weeks the animals were killed and sectioned. The liver, kidney, spleen and stomach were examined. The authors also ran a second series with insoluble manganese phosphate. They also conducted a few human experiments on individuals who had biliary fistulas. Their results were summarized as follows:

1. Manganese is absorbed from the intestinal tract even if the mucosa be intact. However, it may not be possible through this route to produce general intoxication. Most of the manganese is eliminated through the intestinal tract, and very little or none at all through the kidneys.

2. If permanganate be injected subcutaneously it becomes rapidly reduced into an insoluble manganese compound of which only a small fraction is reabsorbed and appears exclusively in the intestine and the feces, but it never appears in the urine. Hence, manganese is taken up by the intestinal tract and is eliminated through the intestinal tract. Furthermore the author believes that manganese has a distinct hematogenous effect which, however, is not so pronounced as that of iron. In the human experiments it was found that some of the manganese was excreted through the bile. Schreiber[14] also wrote a monograph on the absorption of manganese.

On October 7, 1901, Von Jaksch[15] described three cases of diffuse affections of the brain and the spinal cord, which he considered typical cases of multiple sclerosis. These men were all employed as grinders of manganese dioxide in a factory making potassium permanganate. However, Von Jaksch was unwilling to place the blame for the nervous symptoms upon the absorption of manganese. He believed that the inordinate changes in temperature were the direct cause of their disease. But, 11 years later, in his text book on poisoning[16] he admitted these cases had the same symptoms as those described by Couper, Embden and Friedel.

A brief history and a summary of one of his cases is very similar to the case which I am reporting, except for the age, my patient being 10 years older. This patient, 19 years of age had been employed from March, 1900, until December 18, 1900, in the drying of manganese slimes. The dried manganese on large plates was broken up by the workmen wearing wooden shoes. This caused considerable dust which was inhaled by the stampers.[17] In December, 1900, a weakness developed in the patient's leg which made walking difficult. Two weeks later he experienced a sudden trembling and shaking of the arms and legs. About the middle of January he developed a peculiar gait. He felt stiffness in the knees, which made pronounced bending of the legs impossible. He found at the same time that he could walk backwards without falling, and he began to slow up. The physical examination of the heart, lungs, abdomen, development and color of the skin were normal. On February 5, 1901, the patient without cause had a very marked uncontrollable laughter. Long words were uttered with difficulty. He seemed sound mentally, would count from one to 10 forwards and backwards in a monotonous voice. No nystagmus was noted; the pupils were equal and reacted normally. The tongue moved slightly to the right when extended. Fine movements of the fingers were well made, but the movements of the arms were hesitating. The grip of the right hand was strong, but a fine tremor was noticed when the arm was outstretched. The patellar reflexes were increased; no ataxia of the legs; a moderate ankle clonus and Romberg sign, were present. The patient was so unstable he could not stand on one foot alone. There were no sensory disturbances noted. In a week the patient left the clinic, but his speech and gait became progressively worse. On April 20, 1901, he returned to the hospital with a spastic gait. At this time he had a constant motiveless laughter, which did not improve. On April 22, bilateral ankle clonus was present, and

the knee jerks were much more pronounced. On May 3, 1901, he left the hospital with apparently no improvement.

In two cases observed by Von Jaksch, in 1902, no alimentary glycosuria could be found but in the third case he found it to be present. In no case were there disturbances of the visual field. The two cases could not be traced further, but the third was again observed in 1904, and showed a disappearance of the symptoms such as psychic disturbances, impulsive laughter, and weeping. The reflexes were yet somewhat exaggerated and the alimentary glycosuria persisted; of greatest interest, however, was the peculiar spastic gait of the patient. In the latter part of 1906 Von Jaksch saw this patient again. The alimentary glycosuria had subsided and the reflexes were exaggerated, but there were no sensory disturbances. Babinski's sign was negative. The patient stood on a moderately narrow base with open as well as closed eyes; his gait was still peculiar. This gait was of the spastic-paretic type, and he was walking on the metatarsophalangeal joints. He was discharged at the end of 1906.

In 1902, Von Jaksch observed another patient who had been working with manganese for the previous nine months; he had a spastic gait, the tendon reflexes were enormously exaggerated but there was no alimentary glycosuria. The most typical symptom was the gait; with closed eyes he walked much like a drunken person with the only difference that the motions were performed slowly. He improved somewhat, and later served in the army. In 1906 he returned to the clinic, and on examination showed a mask-like expression on his face. His speech was somewhat monotonous, the tendon reflexes were exaggerated, and his gait was of the spastic-paretic type. Von Jaksch was never able to find traces of manganese in urine.

Concerning the etiology of the condition, this author states that originally he had believed this disease was due to some sort of cold trauma, due to the fact that the workman was exposed to enormous differences of temperature. However, he admits that this theory in all likeness is wrong, and now believes that this condition results from introduction of certain manganese compounds into the organism. He noticed that this type of intoxication dated from the time that there had been a kind of brownstone which was very rich in manganous oxide, and was being used mainly for the production of potassium permanganate. The attempt to reproduce the condition in dogs by the manganous oxide remained without success.

On the basis of his experiences, Von Jaksch is convinced that the intoxicating agent is the manganous oxide component of brownstone. He states that from the time manganous oxide has been removed from industry the number of these cases has become nil. As to the pathogenesis of the condition, the author thinks that manganous oxide is taken up by the lungs. This substance combines in the body to form some kind of albuminate which reaches the nervous system. In this sense it acts much like mercury and lead compounds.

Prognosis is favorable as to life, according to Von Jaksch, but some symptoms, particularly those referable to the gait are apt to remain permanently. In agreement with Couper, he has observed that early cases of intoxication may recover. As to treatment, Von Jaksch has used hydrotherapy, galvanization, faradization and special types of exercises. He has frequently used electric currents of high frequency with which he obtained good results.

Finally, Von Jaksch[18] observed a case of a young man who worked for about four years with manganese and who came into his clinic complaining of certain symptoms which were highly suggestive of an incipient manganese intoxication. His gait was spastic, he was standing on a broad base, and he was staggering often. On walking he was standing on the metatarso-phalangeal joints. There was a marked retropulsion. Otherwise there were no disturbances referable to the nervous system. There was a bilateral concentric reduction of the visual fields. Later all of these symptoms subsided spontaneously.

Von Jaksch believes that this case illustrates one of manganophobia, inasmuch as the patient had heard much about manganese poisoning and was so impressed that he developed a neurosis. In both his textbook and in the article in 1907, Von Jaksch gives illustrations of his cases.

EMBDEN[19] observed three cases of so-called "brownstone-grinders' disease" in a mill near Hamburg, and one case in a similar mill in Thureingen. From the observation of these cases he comes to the conclusion that this type of intoxication may be compared to chronic lead poisoning, chronic mercury poisoning and chronic arsenic poisoning, because it represents a type of metallic poisoning which, in common with others, gives rise to typical nervous disturbances. Embden describes the symptomatology and courses of the disease as follows: the disease develops in brownstone workers who are exposed to an atmosphere which is laden with manganese dust particles, usually after several months of continuous exposure. At first there develops an extensive edema of the lower extremities and the nervous disturbances follow within a few weeks' time. Initially there is a sensation of weakness in the back and in the legs and a tendency during the performance of complicated movements to stagger and to run backwards. Later the arms become more or less involved and there appear disturbances of speech and voice. The fully developed condition is pictured as follows:

The patient is apparently in good health but he is unable to perform heavier work. He suffers from paresis involving various regions of the musculature. There is no atrophy. There is an increased tonus which has the tendency to augment or to decrease during repeated active or passive movements. There is a mask-like expression in the face, and the facial musculature shows also an increased tonus. The patient is not able to whistle. The gait is more or less disturbed and uncertain, particularly when the patient attempts to turn. While climbing stairs the patient after having taken a few steps is forced to take two or three steps at a time having the tendency to fall forwards. In complicated movements there is distinct retropulsion, which also ensues if the patient attempts to walk backwards. In walking backwards the patients usually fall. Romberg's sign is usually negative. The tendon reflexes are exaggerated and only in one of the author's cases there was a positive Babinski sign. No anomalies of the skin reflexes were found. In a rigid posture there occurs a coarse tremor of the trunk, extremities and head. There is no intention tremor and no

ataxia. However, in movements which require perfect coordination and a certain amount of force there occurs a distinct action tremor. This can be noticed in actions such as lighting a match, cleaning shoes, etc. In a few cases writing is impaired; usually there is a tremor and a gradual decrease in letter size until finally after a few lines there sets in complete inability to write. The voice is low, as Couper has noticed, uncertain and monotonous. The articulation is somewhat muffled. In one case Embden noticed stammering (Psellismus manganalis, analogous to Psellismus mercurialis). The eye muscles are intact, only one case showed some ptosis. Nystagmus has never been observed. Pupillary reactions are normal, fundus and visual fields are not altered. There is no disturbance of the sensory organs. In the very beginning stage, however, there is some paresthesia in various regions and there is some pain in the back and in the thighs. Psychic functions are not impaired. In four cases observed by Embden there was one case which showed impulsive laughter. Sexual function is not impaired. Of particular interest is the relation which this condition bears to certain neuroses and other nervous disturbances due to other causes. Particularly, its relation to multiple sclerosis is of great interest. As far as treatment is concerned the author advises immediate removal of the workman to some other point. As to the prognosis, Couper has shown with one of his cases that the disease is curable even after six years. Prophylactically, it should be remembered that the work must be carried out in practically dust-free conditions and any workman who shows the slightest disturbances ought to be removed immediately from the place. It seems that a great percentage of workmen are resistant to this type of intoxication, because even after many years of exposure to manganese dioxide they do not develop symptoms.

Friedel and Seiffer reveiwed the most characteristic of the foreign cases. Friedel[20] describes a case which he saw on February 17, 1903: "A copious flow of saliva coming from patient's open mouth. The facial expression is that of a rigid, constant smile. The speech is low, monotonous, disjointed, and almost unintelligible. The gait is heavy and awkward with a slight forward propulsion. During the examination the lips and cheeks began to show fibrillary tremors, and at the same time there was a clonic spasm of the neck muscles which moved the head in slow rhythmic shaking movements about a vertical axis. Repeated examinations gave no evidence of sensory disturbances, no disorders of the bladder or rectum. The Babinski and Romberg signs have been negative. Pupillary reactions, fundi, visual fields, and eye movements have been normal throughout. Only after persistent looking to one side does nystagmus appear. Patient still continued to have uncontrollable laughter and crying."

Seiffer[21] reported a case of a brown ore miller, age 34 years, who had worked three and a half years as a grinder. The early symptoms were night sweats, involuntary movements of the head, fatigue and a sense of stiffness in the legs and arms, increased salivation, low voice, and impulsive laughter. The other symptoms were the same as enumerated by Couper, Von Jaksch and Embden. He found no manganese in the urine, and there were no blood changes. He states that the clinical picture is much like that of multiple sclerosis. However, on careful clinical examination the two conditions may be easily differentiated.

Wagener [17] also briefly reviews cases reported in the past, and continues with the occurrence and therapeutic uses of manganese. The toxicology as described by Kobert, Cahn and Harnack is also given. He collected specimens of urine from mill workers. He came to the conclusion that manganese is absorbed by the intact intestine and appears in small amounts in the urine. The fact that many authors fail to find it in traces in urine is probably due to the fact that the patient was observed too late. He believes that some of the manganese is stored in the body. He concludes that the manganese is absorbed through the gastro-intestinal tract, skin and lungs; that, youthful individuals should not be employed in this line of work; that periodical examinations should be made; and that when any of the early symptoms appear the workman should be promptly removed from the factory.

Neisser[22] reported an inspection of 200 workers in a mosaic factory in Annaberg where brownstone was used extensively for the manufacture of the product. A committee composed of medical experts and health officers visited Chemnitz. The first case he reports is that of a color mixer and siever who had worked interruptedly for 13 years and who complained of fatigue, weakness, headache, vertigo, a sensation of fainting, pains in the leg, arms and back, anorexia, constipation and loosening of the teeth. The committee thought the gastro-intestinal system was the portal of entry of the poison. The second case was that of a woman who complained of neuritis and paralysis of the arms for which she was compelled to stay away from work for 30 days. This did not appear as a true case of manganese poisoning. All the workers in this plant had a pale appearance. It is of interest to note that only two out of the 200 had complaints.

IN 1909, Oppenheimer[23] reports finding manganese in blood, milk, bone, and with traces in the brain. In the ash of pancreatic tissue as much as 2.2% to 2.5% mm.

Friedel[24] reviews the history of chronic manganese intoxication and a case having symptoms typical of this condition, caused by inhaling manganese dust over a period of a few months. Internal rotation of the legs and almost immovable ankle points are features added to previous reports. The intelligence of the patient was not affected. Prognosis he reported as unfavorable.

Von Jaksch[25], in 1910, described all the symptoms of manganese poisoning, giving pathology, prognosis, diagnosis, therapy and anatomical findings. Wilson[26] described progressive lenticular degeneration associated with cirrhosis of the liver without mentioning manganese poisoning. Bertrand and Medigreceanu[27] worked with four rabbits each of which received a subcutaneous manganese sulphate injection in 24-hour intervals. Under the skin of an animal it diffuses rapidly into the organism. A small quantity of the manganese passes through the kidney and appears in the urine. Its elimination is effected also through the liver and bile, and the mucosa of the digestive tract, both of the stomach and intestines.

Stocker[28] describes a progressive lenticular degeneration in a male patient 21 years old. The etiology of the case is not known, but the clinical picture resembles manganese poisoning. The differential diagnosis is not easy between the two con-

ditions. The onset of progressive lenticular degeneration is in adolescence and there is a definite etiology. Stocker comes to the conclusion that progressive lenticular degeneration in all probability is caused by a toxic agent; it seems that the basal ganglia have a special affinity for certain persons of which manganese oxide probably offers the best example. He thinks that there is a familiar predisposition toward disturbances referable to the basal ganglia especially the lenticular nucleus even if the toxic etiology is assumed to be correct.

Seelert[29] gave a review of Friedel's original findings in 1903, also the condition of the patient as he found him in 1913. This author states that about 15 patients had been described in the literature. The author described one case, observed also by Friedel and Sieffer in 1903, in which the patient had been presented to the Society for Psychiatry and Nervous Diseases in Berlin. The usual symptom complex is given, and the findings of Friedel who gave the early picture of the condition. The description of the condition is essentially identical as described elsewhere. The author has not observed any psychiatric disturbances. There was no alimentary glycosuria as described by Von Jaksch. He states that the symptom complex suggests a multiple sclerosis. However, he is not able to give any information concerning the anatomic-pathologic features of the condition. But he suspects that the large cerebral ganglia are somehow involved in the process. He refers to Stocker and Wilson, who described the symptom complex of lenticular degeneration associated with cirrhosis of the liver, which bears a striking similarity to the symptom complex observed in chronic manganese poisoning. He states that no liver involvement was noted in this patient. In 1913 Seelert saw the patient described by Friedel, reporting a striking rigidity of the muscles throughout the entire body; an uncontrollable laughter; and frequently low weeping without any provocation; retropulsion was much stronger than propulsion; the feet touched the floor only with the region of the metatarso-phalangeal joint; in walking, the legs were rotated inwards; the movements of the fingers and arms were awkward; there was no ataxia and no intention tremor; though a quick tremor of the hands was present; the symptoms can be confused with multiple sclerosis; with an occupational history of working in manganese, stiffness of the musculature, with the secondary symptomatology retropulsion, defect of speech, aggravation of active and passive flexibility; the peculiar gait described by Von Jaksch with a lack of sensory disturbances and no involvement of bladder and rectum.

Chop in 1913[30] wrote a monograph on chronic manganese poisoning, which I was unable to procure.

Casamajor[31] in 1913 reported, for the first time in America, an unusual form of mineral poisoning affecting the nervous system of nine men. The men were separating iron from manganese by the use of an electromagnet. The air which the workmen breathed was laden with a fine gray dust. After a man had worked in the dust for a length of time varying from six months to three years he began to notice difficulty in walking, the main feature of which was inability to walk downhill slowly. On the level ground and uphill the patient had no trouble, but when once started to walk down an incline he went faster until he had to run to keep himself from falling forward. He was unable to stop, and had to run with ever-increasing speed until he ran against some object which stopped him, or until he fell down. Retropulsion developed together with propulsion and the patient found himself unsteady on his feet whenever he wanted to move. Walking backwards was impossible, for as soon as the man tried it, he fell backwards or sat down. With the onset, pain and stiffness in the legs were noticed in four cases, insomnia in one, and difficulty in writing in one. About half of the patients had defective hearing not due to middle ear disease. The speech was slightly slurring in some cases. The face in all men has somewhat of the mask-like character of paralysis agitans. There was more or less tendency to impulsive laughter. Tremor of the tongue was common, as was also a fine static tremor of the hands. Laboratory work offered little aid in the diagnosis. The urine and spinal fluid were negative. The course of the disease was chronic, and all of these nine cases improved but not one recovered. One case came to autopsy. Grossly the brain and cord showed nothing abnormal. There was no arteriosclerosis of the large vessels or any areas of softening. From superficial inspection of the brain stem blocks, before embedding, it appeared that the perivascular spaces of the lenticular nuclei and the outer part of the optic thalami were considerably larger than normal. There were no changes in the kidneys, liver or spleen.

ACCORDING to Rambousek[32] manganese and its compounds are dangerous when absorbed into the system as dust. However, this statement has been challenged, as the presence or absence of protoxids may determine the relative toxicity of the dust. It has been shown that ores from the Caucasus containing only a small quantitiy of protoxids are less toxic than those derived from Thuringia and Japan. The uses of manganese are described, and symptoms as given by Von Jaksch are reported.

Casamajor[33], in 1916, discussed the toxicology of manganese and reviewed the literature briefly. He subsequently described the symptom complex. chiefly on 10 cases which he had come under his personal observation. He discussed further the pathology and therapeutic measures applied—arriving at the conclusion, that all remedial agents were failures.

Gairdner[34], in 1918, calls to the attention of the editor of nomenclature of disease of the *Lancet*, the fact that the metal manganese for the first time finds a place among the poisons. He states that the name does not appear in the editions of 1896 and 1906. However, he points out that, in 1894, a young girl died from the fumes emitted from the chimneys of the shops of a blacksmith and plumber during the process of melting the metals they used. These fumes travel a considerable distance, causing a green deposit on wooden fences, trees and the slates and stonework of houses. He voiced the opinion that when injected subcutaneously manganese became poisonous and was cumulative, causing symptoms like lead, zinc and arsenic. Ward[35], in 1918, replied to Gairdner, stating that extensive use of colloidal manganese given by intramuscular injection in furunculosis, acne, eczema, gonorrhea and general systematic infections has shown it to be harmless and non-toxic. He quotes the physiological experiments of Hewlett.

In 1919, Edsall and Drinker published "The Clinical Aspects of Chronic Manganese Poison-

ing,"[36] reviewing the literature, presenting one case, and discussing briefly the etiology and pathology of the poisoning, mainly on the reports of Wilson and Stocker. Laboratory examinations failed to assist in a diagnosis. While men are at work grinding ores, manganese can be found in the urine.

In the same year, Edsall, Wilbur and Drinker[37] published an excellent review of the occurrence, course and prevention of manganese poisoning, based upon the previous literature and 38 new cases, all of which, with one exception, had occurred in the same mill referred to by Casamajor. The crude ore from this mill contained 9% manganese combined as oxides and silicates. Arsenic was absent in this ore, and lead was present only in minute traces. One patient was affected after one and a half months of work, which is the shortest time reported by any authority, while four months and five days exposure to a dusty manganese atmosphere was the shortest time previously noted for crippling of the workman.

Kober[38] records a case of a workman totally incapacitated for four years who was reported to the Ohio Sate Department of Health in 1920. He had worked in manganese for two years. His successor at the mill began to show similar symptoms, but was removed before serious damage occurred.

IN 1920, Sato[39] reported the first case of chronic manganese intoxication in Japan. Conredi[40], in 1930, reported the results of the pharmacological research and therapeutic observations on manganese. Reiman and Minot[41] wrote an excellent paper on the absorption, distribution and excretion of ingested manganese in the form of oxides and silicates. The extent to which swallowing a manganese containing ore involves a possible risk depends primarily upon its solubility in the gastric juice. This point was tested with franklinite (a mixed oxide of manganese, iron and zinc), rhodenite (when pure also called manganese silicate; in the sample employed calcite was the chief impurity), and manganese dioxide—representative manganese ores. One gram of various ores, ground to 200 mesh, was incubated for different lengths of time in 50 cc. of fasting stomach contents obtained in the usual manner from hospital patients. In one case, in order to approach more nearly to normal conditions, C. K. D. took eight grams of franklinite with a test meal which was removed and the stomach washed after 30 minutes. Filtration and analysis were done without incubation. The complete removal of the finely divided dust from a water suspension by filtration proved difficult. The filter finally used was repeatedly proved to be efficient and consisted of an alundum thimble which had been previously dipped in collodin and dried. This was incased in a brass chamber and filtration carried out under about 60 pounds pressure. A few typical figures for solubilities found are as follows: 14. 13 mg. of manganese dissolved after 96 hours at 37°C. in juice with total acidity of 74; 5.56 mg. of manganese dissolved after 22 hours at 37°C. in juice with total acidity of 40; 1. 10 mg. of manganese dissolved, in case of test meal (C. K. D.) in contents with total acidity of 21. Unquestionably the ores are soluable in gastric juice to an extent depending upon the acidity and duration of contact, but sufficient under normal conditions so that workmen who continually swallow small amounts of such ore dusts must continually furnish dissolved manganese for absorption into the blood stream. The various ores showed practically the same solubility. As to the effect of ingested manganese ores on the manganese content of the blood, the investigators found—on the basis of blood specimens from employees of the New Jersey Zinc Company, at Franklin, New Jersey, where several manganese poisonings had occurred—that in no case was the concentration unusually high, and that the mean result as well as the variations from the mean were close to the normal figure. (.012 mg. per 100 gm. of blood.) This is not surprising because in the case of the workmen whose bloods have been studied the intake of manganese, though continuous, was very small, and the normal rate of excretion was more than adequate to keep the concentration at a normal level. This led directly to the question whether larger amounts of manganese taken at one dose may not temporarily increase the manganese of the blood to a higher level. Volunteers were chosen, and the subjects in every case drank a water suspension containing 8 gm. of franklinite. In almost every case the manganese concentration increased slightly during the first hour or two and then fell rapidly back to normal. It is apparent that one or more of the following three factors tends to maintain this low level: (1) the rate of solution of the ore by the gastric juice is very slow, or (2) the rate of absorption into the blood stream is equally slow, or (3) the rate of removal from the blood is too rapid to allow any considerable rise of manganese content of the blood. The literature upon the behavior of foreign crystalloids in the blood stream, and experiments carried out by these authors in which soluble manganese has been injected into the blood stream, cause them to favor the last possibility as a controlling factor, since a few minutes after a considerable intravenous injection of manganese chloride the manganese content of the blood is practically normal. Whatever the controlling factor, since identical results have repeatedly been obtained in the same subject and varying results with different individuals, it is evident that in some subjects the manganese content of the blood can be temporarily doubled by ingestion of manganese compounds while in others the concentration remains constant. It seems probable that individuals falling into the first group would be more susceptible to manganese poisoning than those in whom the concentration does not rise. Regarding the question of elimination of manganese, the rapid return of the blood to a normal manganese content raises the question of its mechanism. Wichert, Kobert, Cahn, and Harnack have stated that manganese is excreted in the bile and intestine, but give very little analytical data. On the basis of carefully conducted human experiments it was found that, like many of the heavy metals, manganese is excreted chiefly by way of the intestines. In no case was there more than a trace found in the urine, while approximately complete recovery was made in the feces, a finding which has been repeatedly corroborated by later work on both men and animals. Following out the suggestion that manganese is eliminated from the blood stream in the bile, the occurrence of the metal in this fluid was studied after feeding franklinite to three persons with biliary fistulas. All the subjects were hospital patients, with constant drainage of bile duct operations.

Control analyses before giving the ore show that the normal manganese content of the bile is very

low, while, after eating the ore, it was very markedly increased. The results do show that the bile is at least one important path of elimination of ingested and absorbed manganese. Even prolonged feeding of large amounts of manganese ore to dogs failed to produce significant changes in manganese content of the blood and tissues or to cause any pathological symptoms. Manganese ores are thus very non-toxic, and in order to produce symptoms of poisoning they must be ingested by individuals who are peculiarly susceptible. Clinical experience has shown that such persons are extremely rare.

Lund, Shaw and Drinker,[42] report (1921) the quantitative distribution of manganese dioxide particles one hour after intravenous injection to the dog, rabbit, guinea-pig, rat, chicken, and turtle. This distribution is remarkably constant for all the animals tested except the cat, in which the injected material is practically equally divided between the lungs and liver. In the other animals the liver performs the main share of the work, and in the cat it has been shown that the liver after 12 hours accumulates the manganese which was formerly deposited in the lungs. The results are in harmony with experiments in which bacterial suspensions are employed for injection, and confirm the suggestion previously made that, in the first handling of foreign particulate material, the animal behaves similarly whether protein or inorganic substances are used.

Lewy and Tiefenbach,[43] briefly review (1921) the literature of chronic manganese intoxication and briefly describe the clinical picture of this condition.

IN ORDER to study the effects of this element on the central nervous system these authors conducted a series of experiments on rabbits. These animals were fed with brownstone, which was mixed with the food; after an incubation period of from two to three months the animals suddenly lost considerable weight and showed characteristic and constant symptoms; among the most characteristic features of this symptoms complex was a peculiar waxy flexibility particularly of the hind legs; the leg could be put in any position; even if this position was most uncomfortable for the animal, it kept the leg in the position it was placed. The pathological findings in the brain include the following: The lesions seem to have special sites of predilection; thus in descending order of frequency they are found in the corpus striatum, in the cerebral cortex, hippocampus and anterior corpora quadrigemina. The lesions involve mostly the gray substance, but the white substance is also somewhat involved. Occasionally there are also foci outside of these sites of predilection, as, for instance, in the spinal cord. The ganglion cells of the cortex show some marked changes. In some areas the diseased cells are densely distributed, in other areas they are farther apart. The lesions are mostly marked in the third layer. Particularly around those vessels which are mostly damaged, the parenchyma shows the most striking changes. In those areas where the parenchyma cells are scarce there is a marked glial proliferation. The satellite cells in acute cases assume an ameboid form, and some of the glial cells show branched processes. The much larger multipolar cells of the corpus striatum show processes which are visible over a long range, the Nissl bodies are shifted toward the border of the cell and they lose their characteristic arrangement within the cell. The entire cell body assumes a homogenous dark appearance, and the nucleus becomes large and dark, and its boundary becomes finally completely obliterated. These ganglion cells are surrounded by numerous satellite cells, as can be seen also in normal preparations. Another characteristic change in this area is the liquefaction of the ganglion cells and their nuclei; this change perhaps bears some relation to the alterations in the blood vessels. In this process the nuclear membrane loses its folds, the nucleolus enlarges and is surrounded by masses of chromatin. The cytoplasm becomes rarified, and many vacuoles may be seen within the cell body. The satellite cells of these ganglion cells have become ameboid. The ganglion cells of the lamina pyramidalis of the hippocampus (cornu ammonis) show sclerotic changes, their processes can be stained over a long distance. There is no glial reaction in this area. The large multipolar ganglion cells do not seem to be very much affected as long as there are no inflammatory foci in this region. But the nuclei of the glial elements are increased in number, and in various places show a very abundant plasma. The nerve cells of the central gray in the brain stem, in the corpora quadri-gemina and in the anterior horn of the spinal cord are affected more or less in the same way. In three cases these investigators have observed changes in the blood vessels which represent all the various forms of proliferation; particularly in the corpus striatum there were seen the formations of new blood vessels, and the endothelial cells showed numerous mitotic figures. In some cases the intima was found to be necrotic with the formation of hyaline thrombi which sometimes were seen to invade the entire lumen of the vessel. Those vessels which have undergone changes are found to be surrounded by glial element which give rise to fibers. In one animal there was found an infiltration with lymphocytes and plasma cells; thus in the corpus striatum there were found, surrounded by some larger vessels, distinct encephalitic foci. The center of such a focus of inflammation is filled out by a necrotic mass which is immediately surrounded with larger epithelioid cells which, in turn, are surrounded by numerous lymphocytes and plasma cells. In older foci there could be seen fibrotic and proliferative changes. In this animal, foci of inflammation have been found also in the cortex. By applying a bacterial stain it has been found that these foci contain large gram-positive rods of the Pasteurella type. The author thinks that the introduction of the poison, which has a predilection for the cerebral areas mentioned above, causes them to become the sites of a lowered resistance; the saprophytic pasteurella of the rabbit, which in normal conditions is unable to invade the organism, settles down in these sites of lowered resistance where it gains in virulence thus giving rise to the inflammatory changes.

Davis and Huey[44] describe (1921) two cases of chronic manganese poisoning which occurred in steel workers who were engaged in handling manganese in the Bessemer process of making steel. These authors describe this process briefly, in order to show the environment in which the

patient worked. One of the patients was an Italian workman, 25 years of age, who was first examined February 1, 1916. The history indicated that he began to be sleepy and indifferent in October, 1915. The symptoms described are similar to those previously given. The feces and urine failed to show traces of manganese. Wassermann test on the patient's blood was negative. Blood count showed: hemoglobin, 85%, white cells, 7,250, red cells, 5,640,000. The authors think that the unusually high erythrocyte count could be explained by a mild form of carbon monoxide fumes. The second case presents almost identical date and concerns an Hungarian workman who for a certain period of time had worked at the electric furnace and had noticed a gradual decrease of his energy and mental acuteness, so that the superintendent thought he was becoming lazy. He had no dizziness, but complained of numbness of his fingers and toes. About this time his speech became muffled and indistinct. The examination of the blood gave a hemoglobin of 90%, leukocytes 8.000, erythrocytes 6,400,000. The high red cell count, the authors think, was a result of a mild carbon monoxide poisoning as in the previous case. A chemical analysis of the urine and stool failed to show the presence of manganese.

McCord[45] speaks briefly of the case reported by Davis and Huey, adding that this is a new disease to add to our list of occupational diseases. Wilson,[46] in 1922, reviews the literature and reports of a case of a man 31 years of age who had worked in Manganese for four years. He had disturbances in gait, arms would go into a spasm, felt depressed and had crying spells. His face was flushed, had a mask like appearance, he perspired freely, had a slow monotonous voice, walked on a broad base, showed propulsion and lateropulsion and gave symptoms of basal ganglion involvement.

Charles[47] reported three cases of manganese poisoning in 1922. He never saw any tendency to weeping or an expression of despondency, but excessive smiling without any cause is very common. Propulsion or lateropulsion may be present, but retropulsion is more frequent. Charles thinks this is almost constant in advanced cases. He found no atrophy, and no alteration in the electrical reactions of the muscles. Romberg sign was not constant; salivation may be increased; sweating, particularly at night, occurred not infrequently. He quotes G. Marshall Findlay: "that manganese chloride, if injected subcutaneously into animals, was extremely toxic to the parenchyma of the liver and kidneys, and that, with small repeated doses this toxic action was chiefly exerted on the liver. He produced cirrhosis of the liver in rabbits, rats and quinea pigs by subcutaneous injection of manganous chloride, and also obtained the same condition by oral administration in rats. The type of cirrhosis was definitely monolobular, and he pointed out that the clue to this action of manganous chloride on the liver, might possibly be due to the fact that manganese salts are largely excreted by the bile."

Stanley Barnes and Weston Hurst are also quoted as having described a series of four cases of hepato-lenticular degeneration in one family, which seems to prove conclusively that the nervous symptoms never occur until the liver has become grossly diseased. They conclude that the cirrhosis in this disease is the result of a series of attacks of acute hepatitis, and that some toxin is poured into the tissues intermittently, and further, that this toxin has a selective action on the lenticular nucleus.

With this in mind Charles gave his patients liver. The symptoms were ameliorated while the patient continued the liver treatment. However, he has not cured any case by the liver method. The author continues to give the detailed description of each of the seven cases which came under his observation. As a safeguard, and to furnish controls, one case of postencephalitic Parkinsonism and two of paralysis agitans were treated by the same method, at the same time, in the same wards. None of these manifested any amelioration of their trouble. Two cases of Sydenham's chorea were also treated by feeding with raw liver, without any obvious effect. In the two cases of paralysis agitans the levulose test revealed no evidence of hepatic inefficiency.

Drinker, Shaw and Drinker,[48] in 1923, report the deposition and subsequent course of particulate material, (manganese dioxide and manganese meta-silicate) administered intravenously to cats and rabbits.

THIS article may be summarized as follows:

1. Suspensions of manganese dioxide given intravenously to the cat are virtually eliminated from the body in one week (168 hours).
2. As this process of removal goes on, there is a transfer of manganese from lungs to liver.
3. Similar suspensions of manganese dioxide given intravenously to rabbits are excreted more rapidly, owing to the fact that in these animals the lungs are not involved and the whole process of removal from the blood is carried out by the liver.
4. A method for the quantitative determination of rhodonite (manganese meta-silicate) is given. (The same as used by Reiman and Minot[41]).
5. Three different suspensions of rhodonite, varying in size of the largest particles from 1.3 to 2 micra, when injected intravenously into cats show a greater tendency toward arrest in the liver as the size of the individual particles becomes smaller.
6. When rhodonite suspensions containing particles averaging 1.1 micron in diameter are injected intravenously into cats, the primary sites of depositions are similar to those observed for manganese dioxide suspensions similarly injected; the disappearance of rhodonite is exceedingly slow and is apparently completed in about 50 days following the injection.
7. Rhodonite should prove a suitable substance with which to attempt the production of lung fibrosis in cats, through the medium of intravenous injection; manganese dioxide, while a useful agent for studying the immediate site of evocalization of particulate material carried by the blood, lacks permanency after deposition and is consequently unsuitable for studies upon chronic changes induced by foreign substances.

Schwarz[49] gives a short report on blood investigations in manganese workers.

Schwarz and Pagels[50], in 1923, used cats as laboratory animals, feeding brownstone from Thureingen, manganese superoxide, manganic ox-

ide; manganous oxide; these substances were mixed with the food, and in one case were blown into the trachea. In the experiments with brownstone the authors observed at first an increase in the hemoglobin and number of red blood cells; after a few weeks they noticed a decrease of the red blood cells to about one half the normal number and also a considerable decrease in the hemoglobin content. Pure manganese dioxide had a more toxic effect in that it produced a rapidly progressing and fatally ending anemia. The toxicity of the preparations depends upon their content of MnO_2 and degree of solubility. The authors therefore recommend that workers who are exposed to the dust of these substances should be examined periodically and in each case a blood count and hemoglobin determination should be made; any individual who shows an abnormally high or low count should be put under observation and, if necessary, should be removed from manganese dust.

Von Jaksch and Wartenhorst[51] report that, in 1924, as the result of numerous cases of intoxications with manganese, the factory regulations have been so modified that the workers do not any more come in contact with brownstone ore dust containing manganese oxides. The primary symptoms consisting of impulsive laughter and weeping, mask like face and monotonous speech, resemble mostly those observed in encephalopathia postgripposa. In the course of four to six years these symptoms disappear except for retropulsion, propulsion and a characteristic gait described as "rooster's walk." It is interesting to note that this poison (manganese oxide) which has been introduced into the body sometimes decades previously, leads slowly toward a permanent lesion of the brain and spinal cord. Von Jaksch emphasizes that a symptom complex which originally is referable to the extra-pyramidal system may, after sufficient time, lead toward a lesion involving the pyramidal system.

Findlay[52], in 1924, found that subcutaneous injections of large doses of manganous chloride are extremely toxic to the parenchyma of the liver and kidney. In small repeated doses the toxic action is chiefly exerted on the liver. It has been possible to produce by repeated injections of manganese chloride an experimental form of liver cirrhosis in rabbits, rats and guinea pigs, while a similar condition has been brought about in rats by the oral administration of the same salt. In its earliest stages the fibrous overgrowth in the liver is confined to the periphery of the lobules in relation to the portal spaces, the type of cirrhosis being very definitely mono-lobular. In its later stages the freshly formed fibrous tissue invades the whole of the lobules. Jaundice is often present. A possible clue to this action of manganous chloride may be found in the fact that manganese salts are very largely excreted from the body by means of the bile.

Mella,[53] in 1924, gave monkeys, over a period of 14 months, gradually increasing doses of manganese chloride administered intraperitoneally. The monkey at first develops movements which are choreic or choreo-athetoid in type, later passing into a stage of rigidity accompanied by disturbances of motility; then appear fine tremors of the hands with the terminal phalanges extended. As one observes these animals passing into the stage of chronic poisoning, the rigidity and disturbance of motility, i. e. the tendency to propulsion with choreo-athetoid movements, and later contractures of the fingers, it is striking how the symptoms simulate those of human cases now considered to be due to disturbances of the basal ganglia. Another striking fact, and one which is probably of great importance, is that we find pathologic changes in the liver, but no abnormality in any other structure excepting the brain. The author is unable to say whether the liver changes precede the basal ganglion changes or vice versa. The relation between the liver and basal ganglion changes in Wilson's disease has not been worked out. From his study Mella found that the liver is the organ that is most heavily laden with manganese, and that it shows the most conspicuous pathologic changes. The article contains a series of very interesting and instructive illustrations.

Gayle,[54] in 1925, reported six cases of manganese poisoning. He is of the opinion that certain persons are not susceptible to this poison, as many workmen have worked for years without any apparent ill effects. He found manganese in the urine of three of the five specimens of urine examined. For prevention of poisoning, he advocates wearing an approved mask; the installation of showers; and baths at the end of the day's work. Dust collector fans, and more efficient ventilation would lessen the chances of poisoning. The treatment suggested was massage, corrective exercises, warm baths and hydrotherapy.

Barnes and Hurst,[55] in 1926, describe the autopsy findings with reference to the brain and liver of a case of hepato-lenticular degeneration. Examination of the tissue for manganese failed to show values higher than the normal manganese content. Charles[56] gives practically the same material as in the preceding, advocating liver feeding in manganese poisoning.

Ashizawa's case[57] (1927) was that of a man in whom typical manganese poisoning developed while he was working as a manganese dioxide grinder and who died three years after the onset of symptoms due to manganese. Examination of the pathological alterations in a case of chronic manganese poisoning disclosed diffuse changes in the central nervous system, but with a definite degree of local intensification. He found that the changes were greater in the gray than in the white matter, and were most pronounced in the mid-brain. The essential pathologic changes found in this case were, in general, the same as those described by Lewy and Tiefenbach for rabbits, and Mella for monkeys, with experimental manganese poisoning. The marked parenchymatous damage found in the author's case had the same localization as the disease process in paralysis agitans and Wilson's disease, a pseudosclerosis. These conditions present many symptoms similar to chronic manganese intoxication.

Martin,[58] describes acute lesions with death, also chronic lesions with formation of collagen. He speaks of the action on the blood, saying that manganese can be substituted for iron, to bring about an increase in hemoglobin, and an increase in the red cells, which, however, may be followed by anemia. He quotes the work of Schwarz and Pagels, that alcoholism may be a predisposing factor, also that phosphorus and arsenic attack, and so reduce the function of the liver that elimination is impaired.

The author emphasizes the following points:

1. That the hepatic lesions may be due to alcohol, also to phosphorus and arsenic contained in the ore.

2. That the bile and the feces are the main source of elimination. The condition of the liver plays an important role in the intoxication. Clinically and experimentally he has always found nervous and hepatic lesions. In this latter connection he mentions the Charles Hormone Theory of treatment. The author says we must admit that we ignore the ultimate chemical mechanism of the action of manganese on the liver. He asks himself the following questions:

3. Is there any connection with Wilson's disease of hepato-lenticular degeneration?

4. What is the histological picture?

Grünstein and Popowa say the lesions are not due to vascular changes. However, the vascular changes described by Mueller in the gray center nuclei do not seem to occur in the human intoxication by manganese. Contrary to lead intoxication, manganese does not act upon the nervous elements. There are six outstanding symptoms, according to this author, which clinch the diagnosis of manganese poisoning:

1. Gait, with rigidity, hypertonia and incoordination.
2. Mask-like expression of the face.
3. Alterations of voice and speech.
4. Tremors.
5. Sensory disturbances. (Not in the usual sense.)
6. Psychic disturbances.

He dwells to some extent upon the various symptoms as he has outlined. Salmon and Planque believe there is a double etiology in this disease since the ores containing .013% of arsenous acid could cause symptoms of arsenical poisoning. The authors call attention to the frequency of pneumonia in manganese workers as previously mentioned by Fischer, Freise and Schopper. Then they dwell upon the differential diagnosis—multiple sclerosis, post-encephalitis, Parkinsonism and Wilson's disease. The treatment they give is practically the same as previously mentioned. They say that late treatment is always unsuccessful. This disease is not compensible in France. Workmen should be selected with great care in this industry. Those with hepatic inefficiency should be eliminated. Alcohol must be considered as a factor. Workmen in this industry should be watched carefully and removed early, as this is a great aid in treatment. Much of the material in this article is similar to that of Dhers.[84] The authors refer to a thesis of Andre Jude on the contribution to the study on chronic manganese poisoning, also Labeylie and Planque's contribution to the study of manganese poisoning.

Bumke[59] gives a brief description of manganese poisoning. He claims that large doses of thiosulphate give some results. Lotmar[60], in 1928, gives a description of this condition found in manganese poisoning.

Cohen[61], in 1928, reviews the literature and claims that chronic manganese intoxication resembles that of paralysis agitans and epidemic encephalitis. In some cases there is also a participation of spinal cord disturbances (sensory disturbances of sexual functions) particularly in the early stage.

Zangger[62], in 1928, saw a case of manganese poisoning in a varnish worker. He reported sensory disturbances, universal in this type of poisoning.

GRUNSTEIN and Popowa[63], in 1929, briefly summarize the few reports of manganese intoxications, and review the experimental work carried out in the attempt to demonstrate the effect of manganese on the central nervous system. (See Schwarz and Pagels[50].) Four rabbits were given a daily dose of two to three grams of powdered manganese in food per 24 hours over a period of one and a half to four months. In the nervous system they demonstrated a degeneration of the nerve cells of the spinal cord, and a degeneration of the nuclei of the cells of the medulla oblongata and pons, of the Purkinje cells in the cerebellum and of the cells in the region of the corpora quadrigemina, and a degeneration of the optic thalami, caudate nucleus and putamen, globus pallidus and the cortex. These changes were mostly pronounced in the corpus striatum. Of all the cells of this region, the small ones of the nucleus caudatus and putamen were seriously damaged in all four cases, without exception. The large cells, on the other hand, of the nucleus caudatus and putamen were seriously damaged in two of the cases, whereas in the remaining two cases the damage was less. The cells of the globus pallidus were damaged severely in three cases but only slightly in one case. In other words, the small cells of the nucleus caudatus and putamen seemed to be mostly sensitive to manganese, and, in descending order of sensitivity, followed the large cells of these structures and the cells of the globus pallidus. These results thus agree with those obtained by Lewy[43] and Mella[53]. Of the other cell groups, aside from those of the striate bodies, the Purkinje cells of the cerebellum seemed to be the most sensitive to the poison. The glia elements showed a markedly pronounced tendency toward proliferation with neuronophagia. The blood vessels showed a distinct hyperplasia of the cellular elements of the intima and adventitia in all cases, and in one case a markedly pronounced lymphocyte infiltration.

Sato[64], in 1930, reported from experimental work a marked difference between bivalent and trivalent manganese. The bivalent ion is two and one half or three times more toxic than the trivalent ion as far as the lethal dose is concerned, and 10 times stronger in causing a drop in blood pressure. The ores in Japan which cause the most trouble are those which contain 70% to 80% or more of manganese dioxide. In studying the effects of manganese on the respiration of the tissues, the author comes to the conclusion that manganese stimulates tissue respiration, provided that only a certain amount be employed; if this given amount is exceeded a contrary effect will be obtained. A part of the physiological action of the manganese found in the body is to act as a catalyzer for oxidation in association with the iron.

Schopper[65], in 1930, discusses pneumonia in brown ore miners. The frequency of fatally ending cases of pneumonia in brown ore miners caused many physicians to incriminate this substance as to specific toxic action. Schopper studied this problem on some autopsy material. Both

cases showed pneumonia, and there could be seen an extensive infiltration of foreign material which, on chemical, analysis proved to be very rich in manganese. This substance had led to indurations throughout the lung tissue. Whether there has been a specific toxic effect of the substance is difficult to state. Further investigations will probably decide this question.

Flintzer[66], (1931) believes there is an inborn or acquired individual predisposition for developing this type of poisoning. He observed two brothers who worked with manganese under practically the same conditions. One of them became ill whereas the other was not affected. Besides this natural disposition there seem to be also predisposing factors, such as alcoholism, excessive smoking, lues, infectious fevers, severe cranial trauma, and feeble-mindedness. Also, poor breathing through the nose has to be thought of as a predisposing factor of greatest significance. The author studied 12 cases of manganese intoxications within three years, and he relates the history of each individual case in detail. The symptom complex observed in these cases is practically the same as has been described by other authors.

Beintker[67], in 1932, reports that workers at the electric arc expose themselves to manganese fumes which are liberated at the electrodes. This author observed one case in a workman 32 years old, who had worked for five years at the electric arc. The symptom complex showed a fine tremor in the extended hands. There was considerable swaying on standing with closed eyes. He had night sweats, and suffered from insomnia. The picture was that of a mild manganese intoxication. Another similar case was reported to the author in a 30-year old man who had worked for 10 years with the electric arc. The electrode in the first case contained 2.18% manganese, in the second 0.2% manganese. The second case showed marked disturbances of speech.

Pignatari[68] (1932) gives the changes in phosphorus content of the brain, in experimental manganese poisoning. In another article the same author writes of the glycemia and lipemia in manganese poisoning. Wilbur[69], in 1932, wrote briefly on manganese poisoning, giving information practically the same as given by previous authors.

Mosheim[70], in 1932, described three cases which came under his observation. These men had been employed in a dry-cell battery plant. One was 31, while the other two were 37 years old. The three men worked in the same factory in a dense cloud of graphite and brownstone particles without adequate protection. Noteworthy in this report of Mosheim is the relatively late appearance of symptoms, two to 12 years after exposure. The first symptoms noted by him were the general weakness, profuse sweating, increased salivation, feeling of stiffness and occasional edema of the lower extremities, dizziness, paresthesia; disturbances in walking with propulsion and retropulsion, micrographia, and disturbances in sexual power. Typical also are the premature appearance of gray hair, digestive disturbances and loss of weight. In the differential diagnosis one must consider encephalitic Parkinsonism. This latter can be ruled out in the absence of encephalitis.

Baader[71], in 1932, (71) observed an interesting case of manganism in a trimmer, who for four consecutive years had been working in Hamburg at the port. In performing the work much dust was produced, which, owing to its content of sulphuric acid, was highly irritating to the buccal and nasal mucosae. The original clinical picture was somewhat confusing because of acute symptoms due to the irritation, (cough, nausea, vomiting.) As time progressed, the symptoms typical of manganese intoxication became very prominent. His condition became much worse, and the clinical picture was typical of a severe manganese poisoning. Sixteen months following his admission to the hospital there were found 3.93mg. of manganese in the feces. According to the author it seems that the unloading of manganese-containing cargoes from a ship is a particularly dangerous occupation, so far as intoxication with this substance is concerned. Baader also thinks that the inhalation of potassium sulphate containing air, and the subsequent inflammatory processes in the lungs, may have predisposed toward severe manganism; in other words manganese was probably absorbed much more rapidly than it would have been were the lung condition normal. Experimental work along these lines should be carried out to learn whether changes in the lungs and respiratory mucosae would influence the absorption rate of inhaled brownstone dust.

Brezina[72], in 1932, discusses types of oxides which give rise to manganese intoxication. The author quotes I. Lowy, discussing symptoms on the basis of the observed histories. Instances are given where the hemoglobin had been reduced to two-thirds of the normal and the erythrocytes increased as much as 9,000,000, per cubic mm. The nervous involvement, according to this author, does not appear earlier than after three months of industrial occupation. Brezina does not add anything new to that which I have included here from previous articles reviewed.

Hilpert[73] discusses the sources of chronic manganese poisoning in the mills in Hamburg, Wernigeroder and Thureingen, and the causes of intoxication. He reviews the work of the German writers, and does not refer to English or American cases. He observed 14 cases. He does not give the individual histories of these cases, but takes them up collectively. Four out of 14 patients were over 44 years of age when the poisoning began. The disease begins on an average of two years after the person has begun to work in the dusty occupation. Some people develop the disease within four or five months, others may not develop it for seven, 10, or even 20 years after industrial employment in a manganese-dusty atmosphere. In the family histories that Hilpert was able to get, he found cancer and pulmonary tuberculosis. Bubarev and Brezina[74] are quoted by Hilpert as believing that manganese-poisoned patients are very prone to have fatal pneumonia. Hilpert himself does not believe that patients are more susceptible to this disease than other people. However, Hilpert does believe that there is a primary damage to the lung or weakness of this organ that predisposes to this condition. Of his 14 cases, one case never showed tremors. One after two years lost the tremor. All the 14 patients showed disturbances of the vegetative system. In the beginning all were very sleepy. Later on some of these patients developed insomnia. One patient had hunger symptoms. Baader reports one having extreme thirst. Only one of his

patients complained of backache. He had three patients who complained of transitory headaches. All 14 of the patients had a peculiar gait, which became more pronounced as the years went by. The tips of the toes seemed to adhere to the ground, and they pushed the legs forward. He noticed the peculiar "rooster's walk" described by Von Jaksch[51] and photographed by Seelert.[29] Motor disturbances of the upper extremities occurred at the height of the disease. All had sexual disturbances. The patients were no longer able to make long movements and were unable to cut or saw wood, or even to clean shoes. All movements had to be made with an apparent push. All movements were jerky.

DIEZ[75] reported a peculiarity of a patient unable to remove a glass from the mouth until he had completely emptied the contents. However, by practicing and counting up to 15 while drinking, he was able to remove the glass. Micrographia was common, and writing became smaller and smaller until the letters were entirely unintelligible. All of his patients had speech disturbances. First, their voice was of a low, hollow, monotonous type. Secondly, they would burst out talking fluently and suddenly stop altogether. There was difficulty in swallowing; it occurred in two out of the 14 cases. One had torticollis spasticus. Two had their knees give away due to sudden loss of tonus as seen in a case of narcolepsy. The disease reminds one of paralysis agitans, with mask-like expression. Psychic disturbances were never observed. Only four out of 14 showed pyramidal signs. Babinski, Oppenheim, Rossolimo, Cloni occurred relatively late. The reflexes were always present. He had two cases of weakening of the convergence reaction. One had a double vision. Examination of the cranial nerves was negative. Sensory disturbances described by Cohen have never been observed.

An examination of these patients showed no pathology referable to the internal organs, and there was no change in the blood picture. The disease may extend for many years, and get progressively worse, particularly as to tonic disturbances, but new symptoms may occur as the pyramidal tract becomes involved. In one case the tremors disappeared. The disease completely incapacitates one for work. The edema is transitory. The night sweats and salivation disappear later in the course of the disease. He has never seen a fatal case of chronic manganese poisoning.

The author concludes that is it typical corticosyndrome problems and is very complicated. The vegetative disturbances can be explained upon the grounds of toxic disturbances referable to the center of vegetative system, and the wall of the third ventricle. Disturbances of sexual function can also be explained upon this basis. Flintzer[66] explained the sexual damage on an organic basis, saying there was damage to the testis. Hilpert, however, believes that it is neurological, since Ashizawa found no pathology in the testis. The involvement of the striatum pallidum may extend to the adjacent pyramidal tract. The factors which decrease the resistence of pallidum striate and make it more susceptible to intoxication are unknown. The theory of vascular damage has been advanced by several authors.

Hilpert[73] says that there are a few important considerations in the differential diagnosis.

First, we must consider (1) mete-encephalitis; however, in this disease the onset is much more insidious. (2) Do not observe a tendency of supination of the legs. (3) The gait is peculiar to manganese. (4) There are no disturbances of sexual functions in mete encephalitis, but there is an increase in libido. (5) The diagnosis is clinched by occupational history.

Second, we must consider paralysis agitans and so-called arterio sclerotic muscular rigidity: (1) These diseases may carry symptoms that are similar to chronic manganese poisoning; one of the differential points is that they start at an older age. (2) If the condition starts early confusion might certainly arise. (3) The history and the onset are the most important points.

Third, multiple sclerosis formerly played an important role, but syndrome is entirely different from that of chronic manganese poisoning.

Fourth, we must consider Wilson's disease, pseudo-sclerosis and torsion dystonia. (1) If the case occurs early in youth, familial history is very important. (2) The onset is insidious in all these diseases; do not see Fleichers corneal ring. (3) There is an impairment of the intelligence. (4) A great resemblance in physiopathological and somatic symptoms between Wilson's disease and manganese poisoning. (5) Attention to the occupation of the patient is of the greatest importance in ruling out these three diseases.

The early picture is so typical that it should offer no difficulty in arriving at a diagnosis. History is not complicated, as all show working in a brownstone industry. Most treatments gave no result. This author uses atropin and scopolamine, but he cannot draw any conclusion about the efficacy.

Salmon and Planque,[76] in 1933, observed four cases. In all four they noticed disturbances in gait, speech defects and mask-like facial expression. They also noticed that not all workmen were susceptible to this condition. In making a complete analysis of the material used by the workers they found that manganese dioxide was present to the extent of 79%, but among the constituents they also found a small percentage of arsenic (0.013%). They came to the conclusion that in reality this condition represented an arsenical intoxication as can be seen from typical symptoms such as weakening and paresis of the inferior extremities, atrophy of the extensor muscles, decrease or even abolition of the reflexes, and peculiar gait. The fact that the old workman who was exposed to the dust for a considerable period of time did not show any symptoms, they attribute to the individual variations in resistance toward arsenical poisoning. They suggest that instead of adopting useless measures to prevent this condition it would be better to add also regular inspections of the factory and periodical examinations of the workmen. The factory should be well ventilated so that no dust may escape, and the workmen should be examined periodically and any subject who proves to be particularly susceptible to arsenical intoxication should be promptly dismissed.

Caso,[77] in 1933, gives the reduced glutathione in blood in experimental manganese poisoning. Chelta,[78] in 1933, observed changes in the phagocytic power in experimental acute and chronic manganese poisoning. Matsumma,[79] in 1933, described the changes in the brain and liver following the ingestion of manganese salts.

Baader,[80] in 1933, reported two more cases com-

ng under his observation of two men, 27 and 31, respectively. The urine in both cases failed to show manganese. The stool gave 7.5 mg. of manganese per 100 grams in the first case, and 2.2 mg. of manganese in the second case. The author furthermore discusses also the significance of pneumonia in brownstone workers, and states that all the cases which are affected by this condition end fatally. Many other investigators have reported that brownstone workers very rarely recover from pneumonia. On autopsy it was found that the lung contained numerous particles of manganese dust, and there was found an extensive connective tissue proliferation in this organ, particularly along the lymphatic channels and along the bronchi. So far as the elimination is concerned, Baader[80] states that manganese is rapidly eliminated through the gastro-intestinal tract; therefore, in cases of intoxication, manganese can always be found in the feces whereas the urine is usually found to be free of manganese, or at most, there may be found only traces. These findings have also been reported experimentally by Handovsky, Schulz and Staemmler.[81]

Freise,[82] in 1933, made extensive observations of 527 workmen: 448 men, 59 women and 20 children. These observations were made over a period of 12 years on miners in the State of Minas Geraes, and on stevedores of the port of Rio de Janeiro who handled manganese ores. The symptoms he observed were not typical of manganese, as the chief complaint was a more or less severe gastro-enteritis.

Bickert[83], in 1933, in his first article reviewed the history of manganese, the various types of ores and the methods of manufacture and uses. In a second article, on the basis of statistical data, he comes to the conclusion that in brownstone factories which employ younger workmen only over short periods there occur fewer cases of intoxications as compared with those factories not taking this precaution. Furthermore, it seems that the hygienic conditions have improved since 1925. The author thinks, however, that many undiagnosed cases of manganese intoxication have been classified among the diseases of the nervous system and of the organs of locomotion.

Dhers[84] gives a general review. Lyon-Caen and Jude[85] observed one case which occurred in an Arabian who had been referred to them and who had worked with manganese dioxide in the production of dry-cell batteries. He showed the usual symptom complex. An investigation was made and the material was found to be composed of manganese dioxide and graphite. Chemical examination of a sample revealed also the presence of silicates, iron, calcium salts, and ammonium salts. Lead, copper and arsenic were not present. In reviewing, subsequently, the literature which was submitted to them by the International Bureau of Labor, they point out that chronic manganese intoxication resembles clinically paralysis agitans, multiple sclerosis and progressive hepatolenticular degeneration. They attempt to give a differential diagnosis of the four conditions. They state that in other countries in which manganese intoxication has been already detected at an earlier period, various prophylactic measures have been instituted. Prophylactically they suggest an adequate ventilation, so that little dust is produced. Furthermore, each workman on admission should be carefully examined and no individual who is affected by hepatic lesions should be admitted. The testing of hepatic function may be very valuable. Also workers who present early symptoms should be dismissed. From a statistical point it would be very valuable to report any disturbance of the nervous system in workmen who are handling manganese compounds. Manganese intoxications are little known in France because few industries employ manganese. Manganese as been substituted therapeutically for iron in the treatment of anemia. Manganese causes at first an increase in the red blood cells, but if the use be prolonged it causes an anemia which may be of hepatic origin.

Leclercq,[86] in 1934, expresses views contrary to those generally accepted: that this poison is introduced into the body through the digestive tract; inhalation seems to play a secondary role, because he thinks that most of the inhaled material is coughed out again. (This of course is not true, as from studies of silicosis we know that some is coughed out, but much is retained when the particles are below 10 micra in size). He quotes Boycott and Cameron[87] that certain hepatic cirrhoses trace their origin to manganese intoxication. The author quotes papers previously mentioned. The symptoms given are practically the same as given before except for signs of neurasthenia and melancholia. Lumbar puncture yielded a fluid which was clear and normal from a histological, biological and chemical point of view. The liver is usually enlarged, but often it seems to be retracted, as in the beginning of an atrophic cirrhosis. This is accompanied by anorexia and gastro-intestinal disturbances. There is often albuminuria. In cases of severe intoxication there is a distinct anemia, as can be seen from examining the blood. In one case the author observed not only a marked decrease in the number of red blood cells and a decrease in the hemoglobin, but also that the red cells showed anisocytosis and polychromatophilia. There was no qualitative or quantitative change of the white blood cells. The coagulability of the blood was found to be normal. Often this condition simulates Wilson's disease, owing to an hepato-lenticular degeneration. But this latter condition is characterized by the fact that it affects adolescence and that it usually runs in families. Often it occurs, as Salmon and Planque have observed, that symptoms due to arsenic poisoning are superimposed on those due to manganese intoxication. In these cases one can observe a paralysis involving the extensor muscles of the leg which accentuates the peculiar gait of the manganese victim. There are also trophic disturbances and cyanosis of the extremities, and often one can also observe plantar hyperkeratosis.

SOURATE[88] observed three cases of chronic manganese intoxication. The first refers to a male patient 27 years of age who had worked in a chemical laboratory and had been exposed to manganese dust for three consecutive years. The second was that of a man 30 years of age who had worked in manganese dust. The third was a man 20 years of age, a brother of the former patient, who had been exposed to manganese dust, but to a lesser extent. The last two patients showed traces of manganese in the urine. Along general lines Sourate thinks that the clinical picture of chronic manganese intoxication is more homogenous as compared with epidemic encephalitis; that in the former condition the cranial nerves are less apt to be involved. Sourate summarizes the condition as fol-

lows: The Parkinsonian syndrome observed in chronic manganese intoxication is characterized by a typical amyostatic symptom complex of rapid development. The symptoms are apt to subside after a certain lapse of time. The disease develops without involvement of the cranial nerves, and its course resembles that of vascular lesions. According to the author's experience, weakening of the sexual power and "rooster's walk" are not pathognomonic for chronic manganese intoxication. The blood picture of chronic manganese intoxication is not specific. Manganese intoxication usually occurs in industrial plants and affects only those workmen who are exposed to manganese dust. Pathologically the condition seems to involve the pallido-striate region and does not extend to the gray matter of the floor of the fourth ventricle. However, the author observed cases with symptoms referable to the pyramidal tract, and he thinks that the original lesion in the globus pallidus may involve also the internal capsule, which would explain the appearance of corresponding pyramidal symptoms.

Owen and Cowen,[89] in 1934, reported four cases of chronic poisoning in men working in the same manganese works. Symptoms appeared in one case after only 8 month's exposure, and in the other three after 2½, 6½, and 8 years. The clinical picture was indistinguishable from chronic post-encephalitic Parkinsonism. Dantin-Gallego[91] described a case of a man 27 years of age, whose work entailed the mixing of pyrolusite (MnO . OH), graphite, and other substances.

Canavan, Cobb and Drinker,[90] in 1934, report a case of manganese poisoning in a man 55 years of age who worked for four years in a mill where manganese-containing fractions were separated from the ores. The mill was extremely dusty; the air carried from 10 to 20 mg. of dust per cubic foot, and this dust contained from 9% to 12% of manganese as soluble oxides and silicates. Thirteen years after leaving the work the man died. The autopsy was confined to the brain. The cerebral hemispheres were equal in length; the cerebellum seemed smaller than it should be in proportion to the brain. There were asymmetrical convolutional patterns in the orbital portions of the frontal lobes, in the temporal lobes and in the occipital tips. The left hemisphere had a simpler type of convolution than the right in the second and third frontal gyri and also in the left parietal gyrus. Frontal sections of the brain showed great atrophy of the cortex on the left, and some indistinctness in the basal ganglia. One slice of the brain which included both striate bodies and optic thalami showed atrophy. One could actually measure the decrease in the size of the basal ganglia, which in different diameters were between 3 mm. and 10 mm. smaller than in a control section. Microscopical examination revealed that the choroid plexus showed cells on the outside of the tufts; they were mostly low and single, but occasionally heaped up. Some tufts showed enlarged vessels; some were thrombosed. The pia mater was definitely thickened. With an elastic stain it was striking to see the disarrangement of the elastic fibrils in many of the vessels. Sections of the cornu ammonis showed a normal pia mater with dilated vessels. The nerve cells stained rather deeply but retained their contour; the lower layers showed cells with satellites; otherwise this region was normal. Sections from the left motor cortex showed fairly well preserved Betz cells. There was an increase of glia in the deeper laminae. Sections from the medulla oblongata were normal, except for slight subependymal gliosis and vacuolation of the ependyma. Sections from the cerebellum showed Purkinje cells which were uneven in placement, swollen and paler than usual; the granule cells were well strained. The cells of the dentate nucleus stained in a range from white lavender to purple; the pale ones were large, the dark ones shrunken. The caudate nucleus was covered with a narrow ventricular ependyma, with vacuoles between the cells, beneath was an accumulation of glia cells packed in down to the level of the vascular bed. Some of the vascular walls were lined with black pigment. The cells of the caudate nucleus were remarkable—there was an excess of pigment in from one half to two thirds of them. They were surrounded by many glia cells, singly and in clusters of from three to seven. No nerve cells with normal Nissl bodies were seen. In the optic thalamus the nerve cells were small and contained some pigment; everywhere glia cells were seen in excess, many as satellites. In the upper part of the structure there were many large cells with pigment, but fewer glia cells around them. In the lenticular nucleus small nerve cells with satellites were seen in the upper part, and there were many glia cells between the nerve cells. There was a marked reduction in the number of nerve cells, so that whole fields of only glia occurred. In the lower part of the section pale but pigmented nerve cells were present, and around these was a marked increase of glia. A comparison with the control specimen showed that most of these observations were abnormal. A comparative cell count was made in the basal cell ganglia to show the number of these cells in each portion. It resulted that there was a marked increase in the number of glia cells in all areas. The authors present some interesting and instructive illustrations.

Von Oettingen,[92] in 1935, has given a very complete review of the pharmacology of manganese. Von Oettingen was able to find only 70 cases of chronic manganese poisoning, whereas I found 136 cases. One of Von Oettingen's cases was Couper's which is an acute case rather than a chronic case of manganese poisoning, from swallowing, with recovery in 24 hours.

Labeylies and Planque,[93] in 1935, observed three cases, and a few cases have been reported to them from others. The symptom complex is described as usual. Inasmuch as the authors have not informed themselves about chronic manganese intoxication until recently, a definite diagnosis originally could not be given. The diagnoses given were either hepatolenticular degeneration or post-encephalitic paralysis agitans. Salmon and Planque, in 1933, analyzed the material and found it contained small amounts of arsenic. They thought that this condition represented an arsenical poisoning. Labeylies and Planque think now, however, that arsenical poisoning is to be excluded inasmuch as no typical symptoms have been observed. However, they think that arsenic may play some kind of adjuvant role. In spite of all kinds of improvements which have been made in France since 1933, in 1934 more cases have been developed. They review briefly the foreign literature. Of the seven cases which the authors have observed altogether, two are completely incapacitated and unable to carry on any work.

In a second article, Lyon-Caen and Jude[94] report that there are very few cases of manganese poisoning in France because of the few industries employing manganese. They had seen only one case of manganese poisoning, reported in the first article. They believe that manganese intoxication occurs through the respiratory and digestive tracts. They quote Handovsky, Schulz and Staemmler, that acute intoxication occurs with paralysis of the central nervous system.

Erben[95] gives natural occurrence and the results of experimental work with manganese.

THE CASE which I want to present,[96] (W. L. B., age 29, sent to me by Dr. H. J. Hyslop, of Freeport, Illinois) entered the Presbyterian Hospital, Chicago, on April 5, 1935. I had examined him several weeks before and his condition was practically the same at this time. He gave a history of having worked in the ore department of a battery company, transporting, loading and handling in various ways 58% manganese in powdered form. He was engaged in this type of work for 14 months, when he noticed that after dumping a wheelbarrow of dust, he stepped back and had difficulty in stopping himself. In a week's time he had become progressively worse. Because of the danger of injuring himself by falling into machinery in the plant, he stopped work and has not worked since. There was practically no change in his condition for six months. Then he noticed if he walked too far he developed a rapid gait, which ended in a fall unless someone was present to assist him. This tendency progressed for about four months, and has remained practically the same since. At the present time he has great difficulty in walking alone, and is afraid to trust himself. This condition is aggravated by fatigue and excitement. In the last five months he has developed a tremor of the right leg. It is a coarse tremor. He can stop it voluntarily by putting his foot on the floor and holding it there. But the tremor begins again when his attention is diverted. There has been no tremor of the hands, head or left leg. He has never developed any sensory disturbances. He claims that he sleeps well, uses tobacco moderately, and does not use alcohol because he finds that it makes his legs worse. The patient was married at the age of 23 years; has one child four years old. Physical examination reveals a well nourished well developed white male lying quietly in bed, not acutely ill. His pupils are wide and equal; react to accommodation, and directly and consensually to light. The fields are normal to gross examination. The fundi are normal. There is no ocular palsy or strabismus. There is a lateral nystagmus at extreme right gaze. There is no obstruction to the nose; no perforated or deviated septum; no discharge. His hearing is unusually acute. He hears a small watch tick 18 inches from his right ear and 12 inches from his left ear. His teeth are in good condition. An upper molar is absent on the right side. The gums are somewhat receded. The tonsils are not enlarged. Posterior pillars injected. The tongue protrudes in midline. There is no atrophy. On the left side of the neck there is an enlarged lymph gland of the size of a hazel nut; the thyroid is not enlarged; there is no rigidity. Chest is well developed. The excursions are equal and normal. The expansion of the lungs is good. The tactile fremitus is equal and normal; the resonance is equal throughout; the breath sounds are vesicular; there are no rales, no rubs. The heart tones are clear; there are no murmurs or rubs. Abdomen is somewhat resistant; there is no rigidity or tenderness. The neurological examination was made by Drs. Peter Bassoe and H. M. Richter. There were no disturbances referable to the cranial nerves. Reflexes are as follows: The biceps, Hoffman, knee clonus, ankle clonus, Babinski, Oppenheim, Chaddock and Gordon are negative; Triceps, radial, upper abdomen, lower abdomen, patellar and ankle jerk are positive. The touch is unimpaired. He feels pain very acutely. Has a normal reaction to temperature. Proprioception, position unimpaired. Heel to knee—right and left—positive. Finger to finger —a slight ataxia. Motor: Strength of flexors of leg seems a little impaired. Romberg, slightly positive. The patient stands steady for a time, after which he may begin to sway slightly. There is no adiadochokinesis, astereognosis, aphasia. Gait: The patient stands on a wide base and is very unsteady and uncertain of himself. He needs help to walk and moves with a propulsion gait. If asked to step back he takes a few steps and falls into the examiner's arms. At times he walks on his toes. He tends to slide the right foot. The gait appears to be spastic in type. The facial expression is somewhat fixed, and the patient does not talk a great deal. April 7, 1935, the blood count is not significant of anything. Lymphocytes are increased a bit. Monocytes are up slightly. April 9, 1935, the face is definitely expressionless. Voice is a little monotonous. There is a slight tremor of the tongue, he protrudes and retracts the tongue rapidly. There are no motor disturbances in the arms; no rigidity; no adiadochokinesis. Wiggles his fingers well. The strength and movements of the legs are good. There is no perceptive increase in tone in leg musculature. On April 13, 1935, there is no excessive drowsiness. No cramping or feeling of stiffness in the muscles. Examination as before. Biceps and triceps reflexes in the left arm are slightly exaggerated. There are several equivalent responses in the left plantar region. There is a fine tremor of the hands and arms. It is very slight in both legs. The spinal fluid is clear, flows freely, rises to 8 cm. in water and to 18 cm. when both jugulars are compressed; it falls promptly when they are released. There is no intention tremor. April 12, 1935, x-rays show extosis on lower medial edge of right femur, probably congenital. There is no other change. Patient feels well. There is nothing significant in the spinal fluid. Sugar is a bit high, proteins are slightly below normal. April 13, 1935, the patient walks with less difficulty. Patient's temperature from April 5 to April 11, 1935, was from 97.6 to 99. His pulse varied from 64 to 96. His respiration varied from 20 to 24. April 10, 1935, the patient was given the Rose-Bengal liver function test—10 cc. of dye was injected intravenously—5 cc. of blood was withdrawn at two, 10, and 18 minutes later. At 10 minutes 51.5% of dye remained in the blood (55% is normal) and at 18 minutes 27.6% (35% is normal.) Liver function as indicated by this test is excellent. Blood Wassermann was negative. Blood chemistry is normal except for blood cholesterol which is 196.6. April 12, 1935, 940 cc. of urine for manganese is negative. April 13, 1935, 1000 cc. of urine for manganese is negative. April 13, 1935, 1000 cc. of urine for manganese is negative; 72 grams of stool is negative for manganese. Spinal fluid is negative for manganese. Blood contained on admission .035 mgm.

of Mn.; and four months later, .028 mgm. of Mn. per 100 cc. of blood, which is only slightly over the normal.

On September 24, 1935, I again examined the patient and found that he has a positive Babinski on both sides, that there is a lagging of the patellar reflex on the right side. There are no sensory disturbances. He now has greater disturbance in walking than at any time previously. He has propulsion, retropulsion, and lateropulsion. He has an action tremor. When he starts to do something, like dressing himself, the tremor is very marked. When he is not doing anything with his hands, there is no tremor. His pupils at this time are wide and equal and react to accommodation and light. There is a lateral nystagmus at extreme right gaze. There is more of a monotonous tone to his voice than at previous examinations. The mask-like expression is still present. He now drags the right foot more than ever before and walks on the metatarso-phalangeal joints, as the "rooster walk" described by Von Jaksch. The patient is now more unsteady and uncertain of himself than at the previous examinations.

In treatment, the patient was given one ampoule of thiosulphate every three days.

BLOOD EXAMINATION:

Hb%	R.B.C.	W.B.C.	N.	S.M.	T.E.	Stain	Counted cells
90	5,030,000	10,000	56	39	41	W	100

URINALYSIS:

Appearance	Specific Gravity	Rea.	Sug.	Alb.	Bile.	Blood	Sediment.
Straw clear	1.010	Alk.	0	0	0	0	RBC.....0 WBC ....0 Casts ....0 Epith. cells .....0 Debris ..0

BLOOD CONSTITUENTS IN MGMS. PER 100 CC.:

Wasser mann	Cholesterol	Urea N	Uric Acid	Creatinine	Total N.P.N.	Co., Vol. %	Sugar	Chlorides as NaCl
Neg.	196.6	14.0	4.0	1.7	37.6	61.4	100.00	470

CEREBRO-SPINAL FLUID:

Cell count	Nonne	Ross Jones	Wassermann	Lange 1,2,3,4,5,6,7,8,9,10	Sug.	Tot. Pro.
1 WBC	Neg.		Neg.	0 0 0 1 1 1 0 0 0 0	75.4	26.4

Summary

I HAVE found 131 cases of chronic manganese poisoning in the literature up to August, 1935. The one case I am reporting and one other that I have seen, make a total of 133 cases. All of these patients were actively engaged in a dusty atmosphere from one and a half months to 28 years, before symptoms appeared. The crude ore causing the dust contained from 9.5 to 80% MnO_2. In Table 1, I have collected 81 signs, symptoms, or conditions reported by various authors. Some of these signs are duplications and could be placed under one heading. For instance, peculiarities of gait are mentioned by all authors, still some express this again by writing propulsion, retropulsion, spastic gait, etc.

The *first* consideration in arriving at a diagnosis of chronic manganese poisoning would be an occupational history of having worked in manganese for over three months. *Second*, a peculiar gait which occurs in all cases (where gait was mentioned by authors, 85%, and where it was not mentioned specifically other symptoms covered this condition, as propulsion, retropulsion, spastic gait, "rooster's walk," etc.) *Third*, weakness in legs, tremors, 88%. *Fourth*, mask-like face 83%. *Fifth*, impulsive laughter, 70%. *Sixth*, propulsion and retropulsion, 70%; tremors or whole body or extremities, 70%. *Seventh*, monotonous speech, or disturbance of speech, 60%. *Eighth*, fatigue, 52.9%. *Ninth*, disturbance in upper extremities. *Tenth*, increase of tendon reflexes, 50%. *Eleventh*, all reflexes exaggerated, 50%. *Twelfth*, sudden onset, 50%. *Thirteenth*, spastic gait, 42%. *Fourteenth*, pains in legs, arms and back, 36%. *Fifteenth*, no sensory disturbances. *Sixteenth*, micrographia. And *seventeenth*, impulsive weeping, 26%. Disturbances of sexual function, 25%. (This probably would be larger in number, but was neglected or omitted entirely in many histories.) All of the other symptoms occurred less than 25%. Laboratory examination does not aid in the diagnosis unless the examinations are made early or while the patient is still at work. The urine has repeatedly been negative for manganese. Blood normally contains .01 to .02 manganese per 100 grams of blood. Examinations made after the men have been away from the mills for months would not show abnormal amounts of manganese. The blood examinations would show variations according to the time of removal from the dusty atmosphere. The erythrocytes might be over 6,000,000 or less than 5,000,000 per cubic mm. Most of the blood counts and other examinations of the blood have been normal. Lymphocytosis occurred in 33.8% of the cases. The blood pressure is normal. The spinal fluid examinations have all failed to show manganese or other abnormalities from histological, biological, and chemical points of view. The feces have shown small amounts of manganese, 2.2 mgm. to 7.5 mgm. per 100 grams of sample. Part of this might have been from coffee, tea, or food consumed by the individual, as manganese is in all plant and animal tissue. The shortest period of work causing illness occurred in one and a half months and the longest period before the first symptoms occurred was 28 years. Therefore it seems there is no immunity against this poison although some individuals are particularly susceptible to this type of intoxication, since only two or three out of every 200 workmen in manganese mills develop the disease. It seems that most of the cases occur in young men 19 to 30 years of age. Liver involvement did not occur in my case. Seelert and others have reported the same findings. The disease develops without involvement of the cranial nerves. Sensory symptoms have been negative in nearly 100% of the cases. Cohen[61] reports sensory disturbances in the lumbar and sacral regions. New symptoms may appear as the disease progresses. Some cases where drowsiness was reported later developed insomnia. Muscular twitching, or tremors, while reported in 88% of the cases probably was present at some time in 100% of the cases. Chronic manganese intoxication permanently incapacitates one, but does not shorten the span of life. Most authorities state that all cases affected by pneumonia end fatally. From the few postmortem examinations made, the conditions found were very similar to those seen grossly in silicosis, an extensive connective tissue proliferation in the lung particularly along the lymphatic channels and along the bronchi.

This condition often simulates Wilson's disease due to a hepatico-lenticular degeneration. But this latter condition is characterized by the fact that it affects adolescence and that it usually runs in families. The coarseness of the tremor, the early onset of dysarthria and dysphagia with the characteristic mental symptoms and the very rapid course in the acute cases with fever and emaciation, would tend to favor Wilson's disease. In manganese poisoning, the patients always are well nourished, and the disease comes on suddenly.

Manganese intoxication also often simulates Parkinson's disease. But this is a slow progressive ganglia involvement, and states that we should consider manganese among the etiological factors.

Some of the cases represent a typical disturbance referable to the pyramidal system (see Ref. 66 and 80); others to a disturbance in the striate body caused by manganese dust. (See Ref. 5, 15, 16, 18, 19, 25, 36, 43, 44, 46, 52, 54, 57, 63, 66, 71, 73, 88.)

Prophylaxis

OF THE greatest importance in preventing this disease is the total elimination of the manganese dust. Industry was awakened to the silicosis problem when millions of dollars of lawsuits were

Symptom or Sign	No. of Cases	Percent
Total Histories Examined, 68		
Initial symptoms (weakness in legs, tremors, etc.)	60	88
Disturbances in gait	58	85
Mask-like face	57	83
Impulsive laughter	48	70
Pro-and-Retropulsion	48	70
Tremor (either whole body or extremities)	45	66
Speech monotonous	43	63.2
Disturbance in speech	41	60
Fatigue	36	52.9
Disturbance in upper extremities	36	52.9
Sudden onset	34	50
All reflexes exaggerated	34	50
Increase of tendon reflex	32	47
Spastic gait	29	42
Broad base in walking	28	41
Pains in legs, arms, back	25	36
Micrographia	23	33.8
Lymphocytosis	23	33.8
Chronic catarrh of the upper respiratory passages	19	27.9
Impulsive weeping	18	26
Disturbance of sexual function	17	25
Positive Romberg	17	25
Feeble-mindedness (already present)	16	23
Lateropulsion	15	22
Tremor of hands	15	22
Unable to walk down-hill slowly	14	20
Manganese found in blood and urine	13	19
Tremor of tongue	13	19
Somnolence	12	17.6
Laboratory examination negative	11	16
Changes in cerebro-spinal fluid	10	14
Pateller reflex exaggerated	10	14
Positive Babinski	9	13
Increased perspiration	9	13
Tendon reflexes exaggerated	8	11
No atrophy of the muscles	8	11
Slow pulse	8	11
Disturbance in swallowing	8	11
Increased salivation	8	11
Cough	6	8.8
Edema	6	8.8
Euphoria	6	8.8
Headache	6	8.8
Disturbance in convergence (eye)	6	8.8
Slapping Gait	5	7.4
Difficulty in turning around	5	7.4
Ankle clonus	5	7.4
Defective hearing not due to middle ear diseases	5	7.4
Clonus of foot	5	7.4
Loss of appetite	4	5.8
Tremors of fingers	4	5.8
Mental dullness	4	5.8
Dizziness	4	5.8
Inherited nervous disturbance	4	5.8
Rooster walk	4	5.8
Gastro-intestinal disturbances	4	5.8
Insomnia	3	4.2
Alimentary glycosuria	3	4.2
Disturbed intelligence	3	4.2
Constipation	3	4.2
Paresthesia	3	4.2
Abdominal pains	3	4.2
Fainting	2	2.8
Paranoid delusions	2	2.8
Achilles reflexes exaggerated	2	2.8
Pill rolling movements of hands like paralysis agitans	2	2.8
Reaction to light and accommodation normal in all except	2	2.8
Teeth loosened	2	2.8
Numbness of fingers and toes	2	2.8
Tremor of eyelids	2	2.8
Memory impaired	2	2.8
Laziness	2	2.8
Violent temper	1	1.4
Twitching of fingers	1	1.4
Hair falling out	1	1.4
Anemia	1	1.4
Hyperesthesia of the lower extremities	1	1.4
Social attitude	1	1.4
Nystagmus	0	0
Positive Wassermann	0	0
Sensory disturbances	0	0

Taking into consideration the duplication of the signs in the histories:

1. Weakness or languor would be 100%;
2. Tremors 100%;
3. Disturbances of gait 100%;
4. Mask-like face 100%;
5. Disturbances of speech 100%;
6. Propulsion, lateropulsion and retropulsion 100%;
7. Reflexes exaggerated 85%.

The other symptoms occur in practically the percentages as given.

Table No. 1

developing disease occurring in the fifth decade of life and the patients do not have fits of convulsive laughter or weeping. The juvenile form of paralysis agitans would be very difficult to differentiate, unless the occupational history was considered. In all other diseases simulating this intoxication the diagnosis is clinched by an occupational history of working in manganese. Some of the constant symptoms in other diseases are not found occurring to the extent of 25% in Table I. Fritsche[97] discusses a case of oligophrenia in which a patient showed symptoms referable to the basal

started. Industry can prevent or minimize manganese dusty atmospheres and prevent this disease. Masks should be worn, wash rooms provided, and every precaution taken as is now taken for lead poisoning. As the intoxication may occur from gastro-intestinal absorption as well as inhalation, washing of hands and change of clothing should be insisted upon at lunch time. Provisions should be made for a rapid change to other clothes at the lunch hour for those covered with lead, manganese or other harmful dusts.

When an excellent exhaust system was intro-

duced into the Wernigeroder Mill in 1902[73], new cases of chronic manganese poisoning disappeared, while other mills continue to furnish new cases. According to Bommert[98] 31 cases of manganese poisoning in Germany, have been legally compensated since the Compensation Law of February 11, 1929, became effective.

Pathology

THE pathology of chronic manganese intoxication has not been entirely elucidated, since only a few autopsies have been performed. We have had to rely upon the results of animal experimentation.

Casamajor in 1916, performed the first post-mortem upon a patient who had suffered from chronic manganese poisoning, dying from pneumonia. In the kidneys he found an interstitial nephritis and the liver showed an extensive biliary cirrhosis. He found that in the brain there was some degeneration of the longitudinal fibers of the pons. Casamajor probably failed completely in recognizing the true pathology. In 1927, Ashizawa[57] reported an autopsy on a case of chronic manganese poisoning. This report is complicated by other pathology caused by caries of the vertebrae and cerebral involvement not due to manganese intoxication. Changes in the central nervous system were found. Marked alterations were found in the putamen and nucleus caudatus, and the degeneration of the large ganglion cells was more pronounced than that of the small. The thin nerve fibers were lessened significantly in the striatum, but the thick fibers remained normal.

Canavan, Cobb and Drinker[90] report an autopsy of a man 55 years of age who had been working in a mill where manganese containing fractions were separated from the ores. The air inhaled by this man carried 10-20 mg. of dust per cubic foot, with a manganese content of 9-12%. Thirteen years after leaving the mill he died of cardio-renal disease. The autopsy was confined to the brain, as the abdominal post mortem could not be obtained. Frontal sections of the brain showed greater atrophy of the cortex on the left, and some indistinctness in the basal ganglia. One slice of the brain which included both striate bodies and optic thalami showed atrophy. The decrease in size of the basal ganglia were between 3 and 10 mm. smaller than in a control section. Microscopic examination showed that the cells of the choroid plexus were on the outside of the tufts; they were mostly low and single but occasionally heaped up. Some tufts showed enlarged vessels; some were thrombosed. The pia mater was definitely thickened. With an elastic stain it was striking to see the disarrangement of the elastic fibrils in many of the vessels.

Baader, in 1933,[80] reports that an autopsy on a case dying from pneumonia showed that the lung contained numerous particles of manganese dust, and there was found an extensive connective tissue proliferation in this organ particularly along the lymphatic channels and along the bronchi.

Schopper, in 1930,[65] reported two autopsies of cases of pneumonia in brownstone miners. He did not report on the pathology of the brain. The findings were the same as given by Baader.

Boycott and Cameron[87] believe that certain hepatic cirrhosis owe their origin to manganese intoxication.

Matsumma[79] described the changes in the brain and liver following ingestion of manganese salts by experimental animals. The poison has an elective action on the nervous system. Lewy and Tiefenbach[43] working with rabbits, Mella[53] working with monkeys, and Grunstein and Popowa[63] using rabbits report a histological examination of the nervous system. Previous attempts by Kobert and Chop to poison animals with manganese salts failed. Schwarz and Pagels administered daily to cats, certain amounts of manganese which caused a paralysis of the hind legs, but no histological picture of the condition was reported.

Schwarz and Pagels[50], Caso[77], and Chelta[78] report changes in the blood of animals that are not seen in human beings. For instance, Schwarz and Pagels in 1923, reported a rapidly progressing and fatally-ending anemia in cats fed brownstone.

Determination of Mn.

BLOOD or urine is evaporated to dryness in a silica dish in the presence of sodium carbonate and ashed in an electric muffle. Add sulphuric acid until effervescence ceases then a few drops of nitric, (1-3 dilution.) Filter and to the filtrate add five drops of a 2.5% silver nitrate, and 0.3 gram of crystallized potassium persulphate. Place beaker or test tube containing the unknown in warm water. In a few minutes a purple color of permanganate appears. Run a blank on all chemicals and dishes used. Make up to 50 cc. compare with a standard permanganate solution containing .002 to .025 mg. of manganese. (See Sutton Volumetric Analysis, 11th Ed. p. 269, 1924, Bertrand[99], or Reiman and Minot[41], for details.)

Treatment

CHARLES,[56] treated seven cases with liver, obtaining only temporary relief. Electro therapy, hydrotherapy, potassium iodide and sodium thiosulphate have all been tried. There seems to be little hope for recovery no matter what the treatment.

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Dermatoses

THE following is a corrected discussion of Dr. A. J. Lanza's subject: "What Are Occupational Diseases?" (See *Industrial Medicine* for August, 1935, p. 399):

JOSEPH JORDAN ELLER, M.D., Dermatologist, New York City: "Over 60% of the diseases found in industries, excluding traumatic causes, are skin eruptions. There are thousands of possible causes for skin eruptions due to external irritants. The science of allergy has explained many skin eruptions which have heretofore been thought to have been due to some obscure internal origin. It is known that certain persons have an idiosyncrasy for certain chemicals, while other people are peculiarly sensitive to any one of many external irritants. There are persons, who, when entering a new occupation will at once get an eruption on account of contact with the things that he handles there. The skin of this person, if he continues to work, may become 'hardened' and the eruption disappear. This type of person should not be taken off his job at once, but should be allowed to go through the 'hardening stage.'

"Another phenomenon has been observed, as for example, a person may use the same kind of soap for several years and then suddenly become sensitized to it, and every time that person goes back, even after a rest interval, to the original soap, an eruption again occurs. This is a breaking down of immunity of the skin against years of irritation from a particular substance. Another example of this phenomenon is the fact that a barber may work in his occupation for 20 years and then thereafter, every time he comes in contact with the various substances used in his work, he will have a severe eruption.

"Another one of the great problems in industrial dermatoses is the differentiation of such eruptions from ringworm fungus infections, called athlete's foot by the laity. It has been estimated that there are about 10 million people in this country who suffer from a ringworm fungus infection of the

hands, groins or feet. It is believed that persons suffering from ringworm of the feet are more prone to have eruptions on their hands from other irritants. Many times the dermatologist must answer the following questions: Was the ringworm infection of the hands the first eruption, and did it become aggravated by his occupation? Or did the patient have a dermatitis from his occupation which secondarily became infected with a ringworm fungus?

"I have had difficulty in obtaining from commercial companies statistics on the numbers of skin eruptions found as a result of occupation, and also a compilation of the causes. Various industries should keep a careful record of the numbers of skin diseases, what specific chemicals they are due to, the age, sex, whether disability has been temporary or permanent or partial, and how many cases of malingering are found. It is known that the patient can cause a continuance of his skin eruption by rubbing Carbona or benzine in the skin daily, and this has occasionally been observed in persons who wish to prolong their disability.

"Regarding the importance of the findings of metal in the skin of a patient suffering from industrial dermatosis, I am of the opinion that much more study will have to be done on this subject in order to evaluate the findings thus far reported—the reason for this being that practically all persons have various metals in their skins as a result of ingestion of fruits and foods, particularly canned foods, which contain metallic elements. So many people with apparently normal skins show metallic bands with a spectroscope, that it is speculative whether they have any connection with eczematous eruptions."

The Traumatic Fakir

Cases from Personal Experience

By FRANK P. HAMMOND, M.D.
Chicago

IN THIS day when every employed man and woman, including mature citizens everywhere, are cognizant of the states' compensation acts to a more or less degree of understanding (more often less than more), it is a constant experience of industrial surgeons to find that the injured act, infer or even speak of compensation early in the period of treatment—not infrequently at the session of initial attention.

In our experience* there are three main groups of injured in relation to mental reactions with regard to compensation:

1. Those laborers, whether common or craft, who consider accidental injury only as unexpected, an incident troublesome from the standpoint of partially or wholly preventing them from performing their usual labors. This first group expect only proper and efficient treatment of their injuries and are willing and ready to return to work as, and when, the Doctor tells them they may. They exhibit no particular complaint except that the accident may have been avoided, accusing themselves as much as, or more than, something associated with their employment. In our experience this is the largest of the three groups.

2. The second group consists of those individuals who invariably infer and often complain that the accident was in some way their employer's fault. They show from the very beginning that they are willing to prolong the convalescence, with no evident willingness to return to work. At the suggestion of the doctor that the wound is insufficient to cause cessation of their labors they make it plain that they cannot or will not go back to work. Or, again, when convalescence is sufficiently established, in the opinion of the Doctor, that they may return, they complain and refuse to acquiesce. However, in a few days they are back on the job. Is the injured the one and only one to decide when he shall return to his occupation? In our experience the Doctor is not only a better judge, but when he "knows his business" he should also be the only judge.

3. The third group are those who display all the negatives of the second group, plus false and exaggerated complaints from the day of the injury to the day of discharge, in most cases coming back to the office a week to 10 days after discharge reiterating their complaints and attempting to "show" the Doctor why they are not able to resume their work. This third class are those who would rather receive $10 per week in compensation than $20 per week as salary for labors performed. It is quite apparent that many of this class come back to the Doctor's office subsequent to discharge for some kind of treatment. In some this "request" is a subterfuge. They are anticipating arbitration litigation and if treatment is rendered at a *later* date it would extend their compensation time. Before the Arbitration Board it would be assumed (and rightfully so) that if treatment was rendered, treatment was necessary; if necessary the injured's complaints are valid.

This explanation reminds us of an instance in the not far distant past when we were called to testify before the board. The petitioner's attorney checked the dates of our treatments. It appeared that he was somewhat elated when on further questioning we admitted the injured was at our office two weeks later than our last treatment date. "Yes, he was at our office, but only a check-up of his condition was made—no treatment was rendered." Later this point is elucidated.

Another instance was a recovered knee case, coming to the office at an odd hour three weeks after discharge and *insisting* on our night junior assistant "strapping" his knee. To forestall false claim adjustment we noted on his record card the unauthorized circumstance, and recorded "no charge" for the "call." The board ruled that our treatment period did not extend three weeks longer than we testified was necessary.

In our practice it is an established rule to consider *first* the injured man. Not only first with regard to laying all else aside and giving him the necessary and required treatment but *first* when it comes to checking and reporting the end results. It is a constant experience, however, that not infrequently compensation cases have for one reason or another built up in their mind the anticipation of receiving an exhorbitant award. For this reason it is not an uncommon feeling among laymen, less so with the general profession, that industrial men are working for insurance companies.

We want to reiterate what we have said time and time again, never have we been asked by an industrial concern or insurance company to exag-

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* This is the second of a series of three articles by Dr. Hammond. The third will appear in the January number of *Industrial Medicine*.

gerate or misrepresent the case in its favor, *but* we have been asked to "go along," "help me out," "make it strong" by recovered individuals.

A friend in general practice at one time while discussing the end results of a certain compensation case said: "You are working for the insurance company." Our answer was, "We are against false and exaggerated claims. Can you say the same?" He was whipped.

The mental attributes of man are so powerful and influence his desires and actions so strongly that in our experience, in most instances it is best to minimize the extent of injuries to obtain the more rapid recovery and more stable and "permanent" end results. For instance, an incomplete or non-displaced fracture of small bones may be termed a "crack." The treatment is the same, the disability period is the same, the end results are the same but the mental state by this procedure has been much improved. A fracture of one or more vertebral transverse processes is often spoken of as a fracture of the spine or broken back, both of which cause instant and constant alarm with the injured and his relatives. If spoken of as a broken short rib or a broken small bone at the side of the spine the injured is satisfied and his mental state is comparable to all other injuries that recover without permanent disability.

Is this modus operandi consistent with our statement above that the injured man should always have the *first* consideration? It certainly is. We have stressed the importance of obtaining and maintaining a mental optimism in our injured patients that will result in *complete* recovery. Physical recovery in an individual whose mental state is morbid is not complete recovery.

In conclusion let us stress again the importance of treating the mental aspect of the injured person from the very beginning. Keep him hopeful. Encourage a confidence in himself. Drop a word now and again—every time you attend him—emphasizing the gradual improvement that is taking place. Compliment him on his sensible outlook and cooperation. Then as convalescence is well established begin to anticipate by word, the time he will be able to resume his labors, as: "It won't be long now before you can return to work." — "next week," — "few days now," etc. His morbid mentality has recovered concomitantly with his physical, and he returns to work a *cured man*.

When this method is not employed in treating injured employees we point out two inconsistencies: *first*, the doctor tacitly agrees that the injured's opinion is paramount and supplants his own, and, *second*, the doctor is inveigled into rendering treatment over a longer period than is necessary, incidentally increasing his fee. End results of these irregular practices we term "feeding the injured a sympathetic pill coated with gold."

Blood Silicic Acid

IN THE Kaiser Wilhelm Institute in Dortmund and Minister (Westphalia), experiments on silicosis have been conducted for some time*. They were discussed in *J.A.M.A.* February 10, 1934, p. 471. Prof. Heinrich Kraut determined previously that the silicic acid content of the blood of normal persons amounts to from 1% to 3% of the ash. The average value of the silicic anhydride in the blood ash of 60 persons was 1.8%. The limits were 1.5 and 2.1% respectively. Tests on 79 patients with silicosis revealed that their blood ash nearly always had high silicic acid values, on the average 3.5% of silicic anhydride. The values fluctuated between 2.4 and 4.5%. Patients with silicosis thus have an increased silicic acid content of the blood; that is, dissolved silicic anhydride has been absorbed by the organism. To determine the length of time a miner works on rock formations containing silicic acid before these chemical influences on the organism become manifest, the blood of 265 miners was tested for the silicic acid content in connection with the roentgenologic examination, to which they were subjected at regular intervals. The blood ash of these workers has about the same average silicic anhydride content as has that of other persons, namely, 1.9%; but the upper and lower limits are considerably farther apart (3.1 and 1%). In workers without demonstrable silicotic changes, the upper and lower limits of the silicic acid content move farther apart within the first six years of time they have worked on silica rock, and in the following 12 years the movement is primarily upward. In workers who develop a slight silicosis within the first six years, without becoming incapacitated, a great increase becomes noticeable, up to 3.8% of silicic anhydride. In the two subsequent six-year periods, this increase partly subsides again, doubtless because men with high silicic acid content of the blood are eliminated from the group, because of severe silicosis. This is indicated not only by the comparison with the values of those silicosis patients no longer able to work but also by the results of the examinations of miners who have worked on silica rock more than 18 years, in whom the values closely approach once more the upper and lower limits in normal persons. This is the case particularly in workers without signs of silicosis and also in those who already have a slight silicosis. The wide fluctuations prevent as yet the elimination of those who are in danger on the basis of the abnormal silicic acid values of their blood.

* Berlin Correspondent *J.A.M.A.* (February 9, 1935).

Hip Fractures

IN THE discussion of the management of the fractures of the neck of the femur the following points should be definitely stressed:*

1. The mechanism of production of the fracture and the pathologic changes which have taken place in the bony structures and in the soft parts surrounding the head and neck are important.
2. A careful study of the x-ray films in both the anteroposterior and lateral views will lead one to the proper diagnosis as to the position of the fractured ends.
3. Continued immobilization is even more important in fractures of the neck of the femur than in fractures elsewhere.
4. Under certain conditions we must expect failures from both the non-operative and operative methods by reason of a necrosis of the femoral head and subsequent non-union.
5. In the operative management an absolutely aseptic technique must be used in order to avoid post-operative infection. Infection superimposed upon a fracture of the neck and a poorly-nourished head is almost certain to lead to failures.

* *Internat. J. Med. & Surg.* (April, 1935).

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Our Who's Who

ONE of the nicest compliments we can remember was the recent statement of an industrial surgeon of national prominence, who said, apropos in general of *Industrial Medicine* and in particular of our WHO'S WHO now in preparation: "I believe you are sincerely trying to do a *good* job."

As to the former, our magazine, it speaks for itself. We might add an inference from the remark the president of a little midwestern railroad made about his line: "Right now it isn't as long as some, but it's just as wide as any."

Concerning our WHO'S WHO, however, a few words will not be amiss.

Among lawyers, the so-called "law-list" has been well known for many years. Lawyers in various localities are solicited—for a monetary consideration, of course — to become identified with a particular list, whereupon the publication of their names in the book is supposed to bring to them, from other lawyers in other cities, certain business which must be locally handled, and which is "forwarded" to them, for that purpose and for part of the fee. It seems that this principle is now being urged among doctors, especially traumatic surgeons. "Get in our list — or directory — and the insurance companies will then refer their local cases to you. . . . It costs only $....., and think of the business you'll get."

Without comment on the methods used or the ethics involved, we simply state that:

The specialty of industrial medicine and traumatic surgery deserves a real and regular WHO'S WHO of the very highest ethical standards and all that they imply.

Such a work will serve *all* purposes — reference, directory, etc. And it will serve them best in proportion, first, as it is free from any kind of commercial selection, and, second, as that "priceless ingredient" — the essential element of any really *good* job — is put into it, namely, the love of the work.

We are, therefore, preparing *our* WHO'S WHO IN INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY on the following basis:

In form and content, this, our book, will follow the general style of the *standard* WHO'S WHO, with the emphasis upon completeness of biographical information, and, of course, upon identification — full time, part time or as consultant — with the medical, surgical, occupational disease, medicolegal, compensation, hygiene, and insurance problems of industry.

In style of presentation it will express the highest ethical standards, and will conform in all respects to the best of accepted usages.

No biographical sketch can be paid for in any way; no financial commitment of any kind is involved in the selection of any one of the industrial physicians and traumatic surgeons to be included; the book will not contain any advertising of any kind whatsoever; and there is not, and there will not be, any obligation expressed or implied for anyone to spend or invest or contribute, in connection with its preparation or publication — excepting only the time and pains necessary to respond to the request for information which we have sent, and are sending, to our list of industrial physicians and traumatic surgeons without regard to any other fact than that they are M.D.'s and are in, or interested in, industrial medicine and surgery.

We intend to do a *good* job — creditable in every respect to the medical profession and its standards, and to ourselves and our standards. Already we are receiving a very pleasing quality and quantity of cooperation in the matter of information sent in. We aim to make this WHO'S WHO as complete as possible. To that end we are asking the cooperation of every doctor who shall read this: We believe we deserve it. Give it to us.

"The Priceless Ingredient"

SPEAKING, as we do above, about love of one's work, there is no profession in which this "priceless ingredient" is more tangibly evidenced than among doctors. We encounter it regularly in articles submitted for publication, and recognition of it commands our respect. Two articles in this issue are unusually eloquent of it: "Health Diagnosis in Adults" (p. 573) and "Industrial Manganese Poisoning" (p. 582). Both have the unescapable connotation of a vast amount of work behind them, done with the loving care of the constructive mind. Dr. McNally's exhaustive and masterly covering of industrial manganese poisoning is particularly notable in this regard, in addition to its outstanding technical value. Impressed with the inevitability of a tremendous background, we ascertained that many, many months of intensive study and literally hundreds of dollars of expense were Dr. McNally's price of thoroughness. His result is a masterpiece. For the time being, at least, and undoubtedly for a long time to come, it brings the subject completely up to date: "It's all there is; there isn't any more!"

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LORENZ BOHLER has long been internationally famous for his many successful developments in methods for the treatment of all kinds of fractures. The German editions of his book are well known in America, but their use has naturally been restricted by the limitations of language. Even a fair reading knowledge of German is not sufficient for a complete grasp of principles or a reliable understanding of details of technique. Many American surgeons have wished for a complete and reliable English translation of Dr. Bohler's work, and one is now ready. Not only a complete translation of the fourth German edition, but added material, not previously published, from Dr. Bohler's latest experience with Fractures of the Spine, and of the Neck of the Femur. Not a dull machine-like translation by an uncomprehending literary hack, but a work of appreciation done by a man who is himself a well known master-surgeon. Ernest Hey Groves, editor of the British Journal of Surgery, an authority on fractures who deliberately postponed revision of his own excellent book on the treatment of fractures in order to pay tribute to the Viennese master of the art, and make his work better available to all English-speaking surgeons. It has been well said that Dr. Bohler's book reveals he has that quality of genius which consists in the infinite capacity for taking pains. **All of** his painstakingly acquired knowledge and experience is now available to every American orthopedist and general surgeon.

Bohler's methods are not experiments but a carefully compiled and developed system, superior to that commonly in use, covering many years experience with a large number of cases. Many American practitioners have visited his hospital where thirty to forty visiting surgeons from all countries watch his work daily. In fact the Bohler experience and methods have been for several years increasingly used in many American cities with success and satisfaction. In the words of a well-known California surgeon, "These methods, applied in the treatment of injuries result in a lessened disability period, an earlier return to the proper industrial status of the patient, a shortened period of hospitalization, and the least possible impairment of function. The morale and physical well being of the injured are enhanced and economy to society ensues."

No surgeon should rest content until he has a copy of Bohler on his bookshelf for reference, for comparative purposes to aid him in improving his own methods and technique. The book is a liberal education in fracture treatment.

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Industrial Medicine and Surgery

Before You Hire Inquire

AS TIME goes on the relationship between employer and employee tends to intensify in intimacy. Within the memory of the average man changes in the condition which surround employment have ranged through a considerable scale. We are engaged constantly with social and industrial problems out of whose consideration emerges the slow revolution of ideas which form our codes. At present a great deal of emphasis is laid upon social betterment of classes whose welfare, in times past, was left generously to chance. So closely interrelated are the social and the practical aspects of industrial employment that it seems proper to discuss briefly a subject which bears directly upon their relationship and concerns equally employer and employee.

I refer to the system of physical examinations instituted by nearly all of the larger industries. Although many of these have adopted programs of periodic examination during the course of employment as well as made them required preliminaries to employment, it is the latter I wish to stress. The idea—and the more it is considered the more its mutual value to employer and employee becomes apparent—can be summarized in the title of this article. In the blue-sky days the Better Business Bureau warned of exploitation under the legend—"Before You Invest—Investigate." I have paraphrased an excellent slogan and employed it as a signal to beware of another form of exploitation—insidious and costly: Before You Hire—Inquire.

The relationship between employer and employee may be likened to that between foundation and structure. A poor foundation is a great liability. Employees enlisted haphazardly are just as likely liabilities. If an industrial structure is to be sound and endure, whether large or small, it must devote adequate attention to its workers. Many concerns have seen the wisdom of giving comensurate care to personnel. The others should and eventually will institute a system of physical examinations for employees.

This always results in benefits to employer and employee alike. Both are informed fully thereby and prepared to enter an intimate relationship and understanding. The worker who is given a clear bill of health will enter the job confidently with the respect of his employer. In the case of one who shows some condition which restricts his occupational range, the employer is enabled to assign him to a post in which he can give maximum service without detriment to his health.

The need for a system of physical examinations has been emphasized during the past five years of dubious business conditions, for they have witnessed abuses and exploitation. So-called occupational diseases have been invented and magnified to the profit of the unscrupulous. The insurance (casualty) companies have been called upon to carry extra burdens. Every conceivable condition in no way related to industry is being injected daily, and effort made to enforce it into status of compensability, that is, a result of accident which occurred out of and during employment. When such a claim is advanced successfully, of course, the case immediately becomes compensable and adds directly to the burden already imposed upon industry. Compensation rates, based as they are upon the experience of the previous year, increase accordingly.

As we proceed in our industrial relations, the importance of the physical examination, to inquire into conditions which always should be known before a person is accepted into an organization, is emphasized daily. These conditions are numerous, but, for the purpose of this brief article, it will suffice to speak only of the few which occur more frequently and offer the easiest opportunity for exploitation.

Here may be interpolated, appropriately, mention of old injuries or deformities, possibly disregarded or even forgotten by applicants themselves. Information about them is valuable, because, conceivably, they may cause trouble later. Tuberculosis, likewise, comes to light upon complete examination.

Recently there fell under my observation an excellent example of the dangers which may lie within such possibilities. A worker reported for removal of a fragment of steel which had pierced the cornea of one of his eyes. At the hospital where I sent him for localization, a linear skull fracture was discovered, though old and wholly unrelated to the injury under treatment. The fracture was recorded without comment; in fact, the patient did not know it had been observed. Some months later suit was entered against the employer which cited the fracture as a compensable injury sustained at the same time the piece of steel had struck the cornea.

The defense, thanks to our records, won easily. Further investigation, suggested by the suit, developed the interesting fact that damages had been collected three times previously—once from a railroad and twice from insurance companies for a total in excess of $10,000—for the identical injury. Actually the fracture had been incurred years before and while hard fibrous tissue had closed the fissure, the failure of the skull bones to knit themselves remained quite discernible under competent examination. Without the discovery of the true condition, the hoax might have been perpetrated repeatedly upon other employer victims. I pass now to the more common situations to cover which physical examinations are essential.

First of these, and perhaps most important as a class, are herniae. These impairments unquestionably cost insurance companies in America millions of dollars each year. Every effort on their part and that of the compensation commissions is made to combat the situation but without success. In Illinois the law states very clearly that a worker must report and give a definite history of an accident which arises out of or during the course of employment within 15 days in order for it to be accredited compensable.

Difficulty Lies in Proof

IF A HERNIA, for instance, is reported on the sixteenth day, the insurance company can refuse all medical or surgical attention and base its refusal upon the law. The proof, however, that it did not occur within the legal limit is altogether more difficult. It is extremely simple to eliminate these questionable cases which cause increased insurance rates by means of the preliminary physical examination.

The second most important class of conditions is that of varicose veins in the lower extremities. An industrial worker who has extensive varicose veins may strike his leg against the corner of a box or any projection. Such an accident would cause an open wound which is bound to develop into a varicose ulcer. These ulcers are very difficult to control. They result in long periods of disability and often many weeks of hospitalization. Not only do they work hardship upon the employee but they also discommode organizations, and, particularly if the injured person happens to occupy a key position, disruptions of entire departments may follow. If inter-departmental connections are closely drawn, such an accident conceivably might induce a chaotic disturbance throughout an entire industrial concern.

High Blood Pressures

THE third of these predominant classes of conditions which have risen to prominence during these last few years of sub-normal business includes high blood pressure. Just how far blood pressures have been influenced by the depression and the mental strains associated with it, in some cases together with lack of proper nourishment, is variable with the individual. The examining physician must consider them. When he examines a worker who has been out of employment for two or three years he must take into account, also, the psychic effect of a physical examination.

In consequence of thousands of examinations I have recorded an astonishing large number of high blood pressures which range from 160 to 190 systolic in our younger generation.

The important thing which influences a decision in questions of high blood pressure, and which should

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* R. Robinson Duff, M.D., *in Central Manufacturing District Magazine*, June, 1935.

have the special attention of industry, is the relation between systolic and diastolic. We know it is comparatively safe to continue younger employees in service, despite rather high blood pressures, because the walls of their arteries as well as veins are elastic. A boy with a systolic pressure of 180 over a diastolic of 74 gives extremely wide spread of difference. On the other hand, a man aged from 55 to 60, who is discovered to show a systolic of 220 over a diastolic of 130, may be labeled immediately as an extremely bad risk. Why?

Because the lack of spread forces one to recognize, not only the fact of high blood pressure, but also, associated with it, a well advanced case of arteriosclerosis. This condition prevents or destroys, if you please, the elasticity of the vessels and predisposes the individual to cerebral hemorrhages. A cerebral hemorrhage which occurs during the course of a worker's employment, even, let us say, usual work every day, would make the employer responsible. In every case when it can be proved medically that extra effort was occasioned by whatever the employee was engaged in, such a hemorrhage may be traced directly to such effort as a cause and declared compensable.

In the hands of unscrupulous and unethical lawyers who constantly haunt industrial wards of hospitals —and who even thrust their tenacles into the homes of our employees to trap and entice them into signing innocent looking papers which, in fact, confer powers of attorney which enable them to proceed before industrial commissions—high blood pressures are formidable. These lawyers have on call and under control an equal number of doctors, perfectly willing to appear before the commissions, whose testimony under oath is bound to be accepted. This is one reason, and a most cogent one, why great care must be exercised in the selection of personnel which will work as nearly perfectly as is humanly possible.

An industry which builds its staff soundly and predetermines that every employee unit is as nearly fit as obtainable, will be rewarded over a period of years by a tremendous saving in major accidents and lost time, and minimum interference with the smooth operation of the organization as a whole. As time goes on increased thought is given to these physical examinations. By means of them, in addition, records of a worker's fitness physically, his mental attributes, quality of alertness, and general attitude are discovered, and according to the examiner's reports it is possible to assign him intelligently to work in which he is likely to give maximum satisfaction and where he can function to the mutual advantage of himself and his employer. A man who suffers from an organic heart lesion, for example, could be placed in a position where, because of his keen mental capacity, he would be immensely valuable without risk for many years to come. If, on the contrary, the same individual were accepted without examination, he might be engaged as a laborer where he would break down within a comparatively short time and become a costly liability to the industry.

Large industries which manufacture and handle food products almost uniformly have adopted a system of physical examinations. Foundries, in which exist the dust hazards recently publicized widely, now demand complete examinations to include x-rays of worker's chests before employment of applicants will be considered. Great oil companies place all attendants at service stations only upon completion of very careful physical examinations and, in some cases, in conjunction with group insurers whose policies' provisions increase their benefits in terms of years of service.

With regard to the methods of approach by which a company may know its staff and avoid the pitfalls of exploitation, I believe the system devised by one of our large oil companies is perhaps the most eminent. A person who wishes to become an employee must present himself for a complete physical examination for which he must pay out of his own pocket. The examining physician is appointed by the industry, and charges $2 for his services. He gives the prospective employee a receipt for the $2 fee, and the latter turns in this, his receipt, to his prospective employer together with his application and all other information relative to his education, former employments and other subjects as required. If he proves to be a satisfactory employee his $2 fee is returned at the end of six month's service. This method has been found to enlist a high grade personnel which is intelligent, industrious, and keenly alert.

Recently a foundry decided to adopt examination procedure. It now costs $9 per employee to determine desirability of workers for such employment—$2 for a medical and $7 for a complete x-ray examination. The cost, for 125 employees, accordingly, was $1125. This might seem like a large sum, but consider that one cerebral hemorrhage would cost $4700 in compensation should an employee die or be pensioned for life as a result. The initial expenditure thus seen in its true perspective is a strong argument for a sound, competant organization.

In the interest of a strong foundation for any industrial structure, particularly in these days when rackets multiply far more rapidly than do the means to combat them, I cannot stress too urgently the thought: Before You Hire, Inquire.

Sand Blast Helmets

I AM surgeon for a large foundry. In the sand blast operation the men are protected by masks made of hardened rubber. The air is purified by passing through water. The question has been raised as to what is the best method of sterilizing these masks after they have been used by one person and before they are put on by another employee.—M.D., Ohio*

Answer—The U. S. government, in its specifications for sand blast helmets purchased by its various departments, requires that each article be capable of passing either one of the following sterilization tests:

(a) Immersion for ten minutes in a solution of formaldehyde made by placing one part of 40% solution of formaldehyde in nine parts of water, or

(b) Subjection to sterilization by a moist atmosphere of antiseptic gas, preferably formaldehyde, for a period of 10 minutes, at room temperature.

It has been suggested that care should be taken to remove all the formaldehyde from the masks by washing with water before they are placed in use.

* *J.A.M.A.* Queries

An Experiment in Group Medical Care*

DURING 1922 the employees of the Standard Oil Company of Louisiana at their refinery in Baton Rouge had suffered a reduction in income. They felt that the cost of living in the community had not been reduced comparatively to their reduced income and at a meeting of employees, committees were appointed to contact the professional and business men of the city with a view to a reduction in living costs. There had been a material increase in medical fees in the preceding five years and the employees felt justified in asking for a return to the former schedule of fees. Discussion with the medical society however produced no results.

As a result of these discussions an association was formed in 1923, consisting of 1300 employees. An agreement was made with four physicians by which the members paid $1.00 per year which was divided among the four physicians and these physicians contracted to furnish complete medical service to the members and their families at one-half the usual fees. As was to be expected, this arrangement proved unsatisfactory both to the members and physicians and was abandoned after one year.

The members felt, however, that a satisfactory solution could be found and, after a thorough investigation of available plans, it was decided to form an organization supplying complete medical, hospital and nursing service to members and their families. Two thousand members was the goal set for completing the organization, and on April 1, 1924, the association began to function with 2,200 members.

The cost of operating the association was entirely conjectural and the dues were set at $2.00 per month for each member as an experimental base. The right was reserved to make an assessment of $2.00 per member once each quarter. Seven physicians of Baton Rouge were employed on salaries, including a surgeon and an eye, ear, nose and throat specialist. These physicians were on part time and used their own offices in caring for the beneficiaries. In January, 1925, a clinic was established in rented quarters and the physicians, with

* By JAMES M. ADAMS, M.D., Standard Oil Company of Louisiana; presented at the A. M. A. Personnel and Industrial Relations Conference held at the Hotel Schenley, Pittsburgh, Pa., February 6, 7 and 8, 1935, and copyrighted by American Management Association as "Pers. 16," Personnel Series.

the exception of the surgeon, were placed on a full time basis. Finding it necessary to make assessments frequently and wishing to increase the scope of the work, the membership in January, 1928, voted to increase the dues to $3.00 per month with the privilege of making an assessment of $3.00 three times in a calendar year. It has been necessary to make only two such assessments and one was to care for an unusual expense outside the ordinary operation of the association. Dues are not remitted when a member is sick but no deductions are made from the sickness benefits paid by the company.

There was considerable opposition to the association on the part of the local profession, and in 1930 it appeared that the two hospitals in the city might be closed to the association's medical staff. To provide against this contingency stock subscriptions were solicited from the members and $65,000 was collected to build a hospital. Most subscriptions were for $50 and were paid in installments. The goal was $100,000. Before construction was begun, arrangements were made with the hospitals assuring the use of the hospitals by all of the medical staff and the money collected was returned to the subscribers.

During 1928 a change in the personnel of the staff resulted in the selection of a surgeon and medical director on full time. Thus all the staff were on a full time basis. In October, 1931, the Association purchased a residence and converted it into a clinic. This was financed by the formation of a non-profit corporation with an authorized capital of $100,000. Purchase and retention of one or more shares of stock at $20 per share was made a requisite for membership in the association. Payment for the stock was permitted at the rate of $2.50 per month for eight months for each share. Payment is now permitted at the rate of $1.00 per month. Persons whose membership in the association is terminated may be reimbursed at par on the return of their stock at the discretion of the Board of Directors if in their judgment there will result no impairment of capital. No refunds have been made since the clinic building was purchased except to employees leaving the service before their share of stock is paid up. Non-member owners of stock have no rights other than to share equally in the event of liquidation.

During the depression years when the membership reached a minimum, the salaries of the medical staff were reduced and to compensate for the cut, the physicians were permitted to do outside practice. These salaries have since been raised and all physicians are now on full time.

All employees of the Standard Oil Company of Louisiana and affiliated groups are eligible for membership in the association. When the association was first organized a few negroes joined. Most of these have permitted their membership to lapse so that at present the membership is practically all white. The membership rose from the original 2,200 to 2,800 in 1928 but as a result of a reduction in forces at the refinery, dropped to 1,975 in 1932. The present membership is 2,200 which is 85% of the eligible white employees. Members whose services are terminated with the company are privileged to retain their membership provided they have purchased and retained a share of stock and pay their dues promptly. Annuitants and the widows of deceased members are accorded the same rights.

Membership in the organization is entirely voluntary. Dues are the same for all members regardless of their rate of pay or the number of dependents entitled to service. Dues and assessments are collected by payroll deductions authorized by the member.

The affairs of the Association are managed by a board of directors consisting of 11 members elected by the membership for terms of two years. Eight are elected from specified divisions of the employee group and three at large. Five members are elected to the board one year and six the next making the terms of office overlapping. Officers are a president, vice-president and treasurer elected from the directors and a secretary who may or may not be a member of the board. The present secretary is a member of the board and treasurer. All members of the board serve without pay except that their dues are remitted. The secretary receives a salary of $110 a month and has an assistant who is paid $30 a month. The company has taken no part in the operation or control of the Association other than making occasional substantial contributions for special purposes, making payroll deductions and offering every encouragement. There are always one or more of the executives serving as directors on the board.

The medical staff at present consists of a surgeon who is also medical director, an eye, ear, nose and throat specialist and six general practitioners. The surgeons make no house calls except in consultation, and one of the general practitioners does only office work and anaesthesia. In addition there are two graduate nurses who are laboratory and x-ray technicians, and a practical nurse. One of the nurses does the laboratory work and with the x-ray technician operates the physiotherapy department. The x-ray and clinical laboratories and physiotherapy departments are directed by one of the general practitioners. The practical nurse assists the doctors in their offices and the other graduate nurse serves as head nurse and doctor's assistant. A clerk cares for the office records and telephone calls. A caretaker takes night calls and locates the physicians called. He is given living quarters over the garage and paid a small salary. This has proved very advantageous. In addition two porters are employed, one on part time. The physicians are all highly competent men. Five are comparatively young men graduating during the past eight years and all have served hospital interneships. The other three are older practitioners.

Besides the surgeon and eye, ear, nose and throat man who confine themselves to their specialties, several of the others are partial specialists.

One is in charge of the laboratories, another assists in all operations and replaces the surgeon in his absence, another is especially interested in obstetrics and another in pediatrics. Consultations are frequent and staff meetings are held periodically at which papers are read by members or by visitors from New Orleans.

Each physician has one day off duty each week and a two weeks' vacation with pay every year. They are given time off with pay at intervals for attendance at Medical Society meetings and for post graduate work. They are protected by liability insurance limited to $5,000 per case, three cases per year and double indemnity for suits involving x-ray injuries.

Salaries now being paid are:

Medical Director and Surgeon $600 per month.

Eye, ear, nose and throat specialist $425 per month.

One General Practitioner $400 per month.

Three General Practitioners (each) $350 per month.

One General Practitioner (limited service) $140 per month.

They are required to furnish their own cars and their upkeep. This is the only cost they have in connection with their work.

The clinic building was formerly a residence. It is of two stories and a basement. On the lower floor are located the general waiting room, dispensary or drug-store, administrative office of the Medical Director, office of the eye, ear, nose and throat specialist, physical therapy department and x-ray and clinical laboratories. On the second floor is an emergency operating room, genito-urniary treatment room and four suites of offices each consisting of a joint waiting room, consultation room and two examining and treatment rooms. Each suite is used by two physicians and the office hours are staggered so that one to three of the general practitioners are in the building throughout the day. A dispensary is owned and operated in the clinic by a registered pharmacist. No rent is charged and in return the prices of drugs and prescriptions are lower than in the usual drug stores. It is subject to regulation by the board of directors.

Membership provides unlimited medical service, hospital service up to $250 for a single case in either of the local hospitals, and nursing care by graduate nurses when needed. Dentistry, drugs and x-ray examinations are not furnished. Drugs administered in the clinic such as salvarsan and other intravenous medicines are charged for at reduced rates. Ward service is usually provided, but private rooms are provided when necessary for proper care. Hospital service is not furnished for normal labor cases, but the member can have the association physician's care in the hospital by paying the hospital charges. An x-ray department has recently been installed. A small charge sufficient to cover supplies, overhead and depreciation is made for x-ray examinations. In order to reduce the unit cost of these examinations, x-ray examinations are made for non-member employees

THE BULLETIN

of

The American Association of Industrial Physicians and Surgeons

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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of the company at the same reduced rates. No x-ray treatments are given. A proposal is now being made to the membership to collect an assessment of $3.00 to cover cost of examinations which would then be made without charge.

A very few of the members prefer to retain their family physicians rather than use the association physicians. Hospital and nursing care is furnished these members on the advic of their physicians but they are required to pay their doctor. Consultation with outside doctors is paid for when such consultation is deemed necessary by the attending association doctor.

All the above services are furnished to the dependents of members also.

Included have been the wife, children and totally dependent relatives living with the member. A new ruling has just been passed by the board of directors limiting the dependents to the wife, children and parents of the member if totally dependent on him. Boys over 21 are not included whether dependent or not, unless they are attending school or physically incapacitated. Work after school and part time work of minors does not exclude them. The member may obtain the association's benefits for other totally dependent relatives living with him by the payment of $1.00 per month for each dependent.

No sickness benefits are paid by the association. This phase of relief is cared for by the company's liberal benefit plan and by an entirely separate employees' cooperative organization.

The volume of work done fluctuates within comparatively narrow limits. The cause for some of these fluctuations is apparent while others are more or less unaccountable. The following figures for the year 1933 are representative of the work done annually.

Office visits	27,330
House visits	15,272
Hospital visits	8,369
Obstetrical cases	135
Major operations	120
Minor operations	660
Medical cases in hospital	202
Cystoscopies	52
Laboratory examinations	5,797
Physiotherapy treatments	1,529
Special nurses (cases)	235
Nursing days	1,272
Hospital cases	748
Hospital days	4,314

Two hundred eighty-seven of the minor operations were performed in the clinic and the rest in the hospitals. Included in the hospital minor operations are 198 tonsillectomies and adenactomies.

The total house visits and hospital visits were approximately 85 per cent of the number of office calls but the number of cases treated in the clinic was naturally much larger than those treated by outside visits.

Seven hundred forty-eight cases were hospitalized averaging 5.8 days each in the hospital. Eliminating 239 minor cases requiring only one day hospitalization, the average hospital days per case for the remaining 507 cases was 8.5 days.

Special nurses averaged 5.4 days per case requiring the service. It is in this group requiring hospital and nursing service that the burden of payment for medical service individually would have fallen oppressively.

The distribution of the work among the doctors is not entirely uniform, owing to the freedom of choice of physicians. Every effort is made by the staff and directors to equalize the burden without interferring with this very important prerogative.

The cost of operating the association has been kept fairly close to the receipts but a small balance has been accumulated. This amounted in round numbers on January 1, 1934, to $7,700, not including amount invested to protect "frozen" bank accounts in reorganized bank stock. The special stock account stood at $4,800. Frozen bank accounts amounted to $2,800, giving a net liquid balance on the above date of $9,-700, out of which is held $1,100 for unpaid taxes. Statement for 1934 has not been completed but an addition to surplus of $3,500 is indicated. The total receipts for the year 1933 amounted to approximately $80,000, so the amount of surplus accumulated in the 10 years of operation on January 1, 1934, stood at only 11% of the 1933 receipts. This is not a large margin for unforeseen expenses such as would result from an epidemic.

The receipts for 1933 were made up of $76,900 for dues, $2,600 for drugs administered in the clinic, $125 recieved from the company for treating compensable accident cases and the balance from miscellaneous sources.

The expenditures for that year amounting to $78,150 were distributed as follows:

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The American Public Health Association has been operating a Book Service for years. Its customers are loud in their praises of its convenience. If you buy books — any books — perhaps it can be useful to you as well.

The Book Service renders expert advisory help. It will recommend the most generally approved books, old and new, on any public health specialty, and industrial medicine is such a specialty. It will undertake the furnishing — and guarantee satisfaction — of anything from a "Five-Foot Shelf of Health Books" to an entire library.

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Salaries of physicians	$34,800	43.5%
Hospitalization	16,800	21
Special nurses	7,600	9.5
Office and administrative salaries	7,300	9.1
Drugs and medical supplies	3,150	3.9
Medical equipment	500	.6
Outside physicians	300	.4
Depreciation of building and equipment	1,700	2.1
Excess of receipts over expenditures	1,800	2.2
Miscellaneous operating costs, taxes, etc.	6.050	7.7

These figures are all round numbers.

The clinic building cost $40,000, other realestate $10,000 and medical equipment is valued at $8,000. The association is exempt from income tax but pays real estate taxes.

A discount of 15% is allowed by the hospitals from their regular schedule of fees. Special nurses are paid at the prevailing rates.

In 1932 a study of the Association was made by Dr. Rufus Rorem of the Julius Rosenwald Fund and Dr. John H. Musser, Professor of Medicine of Tulane University and recently president of the American College of Physicians. Their report was published by the Rosenwald Fund under the title "Group Payment for Medical Care."* The conclusions of both these gentlemen were favorable to the service being rendered by this Association. Dr. Musser commented favorably on the maintenance of the personal relationship of the physician and patient. Free choice among the five general practitioners is permitted and the physician stands in the same relation to the patient as the "family doctor." Dr. Musser also complimented the association on the high type of doctors on the staff. During the controversy with the local profession the judicial council of the Louisiana State Medical Society declared that the practice done by this association was not unethical.

This plan seems ideal for caring for the medical needs of this particular group. The membership as a whole is highly appreciative of the benefits. Resignations of members are negligible.

The members are paying a fair price for the service which is probably not greatly lower than the average cost per individual in the community. The compensation of nurses and physicians is above that in the community. Many members have received services which would have cost hundreds of dollars if secured in the usual way. They would have had to accept charity from their fellow employees or the community or have been saddled with debts which they could never expect to pay. The total hospital costs would have been paid by 15% of the membership with physicians' services distributed in about the same proportion. Membership in the association has permitted these men to retain their independence and freedom from debts of this character.

The distribution of the cost equally among all members is most advantageous. The employee on a low wage and with a large family needs protection more and is less able to pay than a highly paid workman with no dependents, yet both pay the same dues making the association truly cooperative.

The treatment received is much better than the average for the group paying for service individually. The professional staff is above the average in ability. There is no hesitancy on account of cost in referring cases or calling consultations. The attending physician sees the patient as often as necessary which is more frequently than is usual with private practitioners. Laboratories are freely used, and x-ray examinations are available at a small additional cost. Patients needing the service are hospitalized promptly.

The physicians are more pleasantly situated than in private practice. They are provided with ample facilities and assistance for carrying on their work. They have the advantage of close association and mutual exchange of ideas. They receive fair compensation for their services without the worries of collections and overhead expenses. They have one day free each week and a two weeks' vacation every year with pay — and with it all are able to retain the valuable personal relationship with their patients.

Preventive medicine is practiced freely. Vaccines and serums are administered routinely for prevention. The members consult the physicians for minor ailments that would ordinarily be neglected and thus more serious disabilities are prevented. The use of patent medicines is discouraged.

Sickness rates in the personnel of the refinery have fallen consistently since the organization of the association and we feel that it results in part, from the improved medical service and preventive medicine as practiced by the association.

The board of directors are earnest and enthusiastic. They do not interfere in any way with the medical work of their physicians, and are alert to limit the impositions on the doctors and the association that sometimes occur.

It is my feeling that of the many benefits enjoyed by this group, membership in this association is one of the most important.

Workmen's Compensation Legislation—Occupational Diseases

IF WE quickly turn back the pages of history 13 centuries, to the year 636, we shall find that there ruled over ancient Lombardy a King by the name of Rothari. The Lombards had invaded Italy, and at that time the laws of the Lombards were mainly in the memories of unlettered judges, who only correctly remembered those laws which were frequently administered. It is hard to conceive of this condition in a country which had a scientific system of jurisprudence, a development from the laws of the Twelve Tables to the Digest of Justinian. Consequently, on November

* Thomas N. Bartlett, Manager, Claim Division, Maryland Casualty Company, Member, Advisory Committee on the Occupational Disease Situation, Association of Casualty and Surety Executives; address delivered at the Twenty-Second of Industrial Accident Boards and Commissions Asheville, N. C., September 30-October 3, 1935.

22, 643, King Rothari, who was the first great legislator of his people, at the age of 38, codified the laws of Lombardy in 388 chapters. In these chapters are to be found at least the origin for the basic principles of compensation which I think history records.

King Rothari's edict applied to personal injuries received in brawls, fights and feuds. Incorporated was a list of injuries with the amount of money to be paid for each, covering death, dismemberment, loss of sight, and other specific injuries. In some cases extra amounts were to be paid for disability. All injured parties would not receive the same amount for the same injury. A freeman received the greatest amount. Then the slave and household servant and several other classifications received lesser amounts. There was a provision for medical benefits. Payments were made in the monetary unit of the solidi which has been estimated to equal 12 shillings, or about $2.92. At the time of such estimate, however, a shilling was worth from five to 10 times as much as it is now. The highest benefit, which was for death, was 60 solidi and probably amounted to from $1,200 to $1,500.

The motive was two-fold. First: to fine the offender and thus create revenue for the King's Court. All of the fine was not paid to the injured, but only part, the balance to go to the King. For death, one-half of the fine was paid to the King. Second: Rothari denounced the practice of adhering to the barbaric custom of resorting to a fight, or duel, or single combat to settle disputes. He fostered the wisdom of promoting compromises.

Even though the payments may be considered conservative, yet they may represent a fair comparison of values as between the seventh and twentieth centuries.

Who can venture an opinion as to the effect and influence, even though slow, on the generations which came and went through the centuries? Spanning these centuries to the middle of the nineteenth, we then see the influence no doubt of Rudolph Von Jhering, a graduate of law at Heidelberg, professor of law at several universities, and one of the most renowned jurists of his time. Several of Von Jhering's claims to distinction, according to authoritative commentators, are based upon the fact that he universalized the Roman Law. He was the founder of modern legal realism. He stood out in his treatment of the nature of legal rights by which he established the juristic basis for a social reconstruction of legal institutions. The outstanding work of Von Jhering —"Struggle for Law"—attracted wide attention. It has been published and republished, the last German edition being the 18th, and has been translated into 30 different languages.

Just a short while before Von Jehring's death, in 1892, Germany in 1884, by an act of its Reichstag passed, what has been accredited to Germany in modern times, the first compensation law.

But the first law which provided for compensation "regardless of fault", while though perhaps somewhat crude in the light of present experience, was that of Switzerland in 1877. This law imposed upon employers responsbility for diseases which specifically and conclusively grew out of certain occupations where poisons or harmful substances were used. Later on in the Swiss law poisons and harmful substances were required to be listed. This list has been gradually enlarged and still is in use at the present time.

It was not until within the past 10 years that Germany extended the "Accident Insurance Law", making compensable as if accidents had occurred, several specified miners' diseases and diseases caused by lead, and a number of other substances, or by radioactive rays. The Federal Council was previously empowered to provide compensation for such occupational diseases as they should specify, but the Council was not apparently convinced of the wisdom of exercising that power. It was not until 1925 that the Minister of Labor, who succeeded the Federal Council, issued the order to make compensable the diseases above mentioned.

In the meantime, Great Britain revised its original Workmen's Compensation Law of 1897, and in 1906 was the first country to follow the example of Switzerland in providing compensation for certain diseases named by schedule.

Following Great Britain's example, practically all other European countries extended their compensation laws to grant coverage for diseases by a schedule, and generally had special provisions differing from those applicable to accidents. Then later followed, comparatively recently, the compensation laws of South America,

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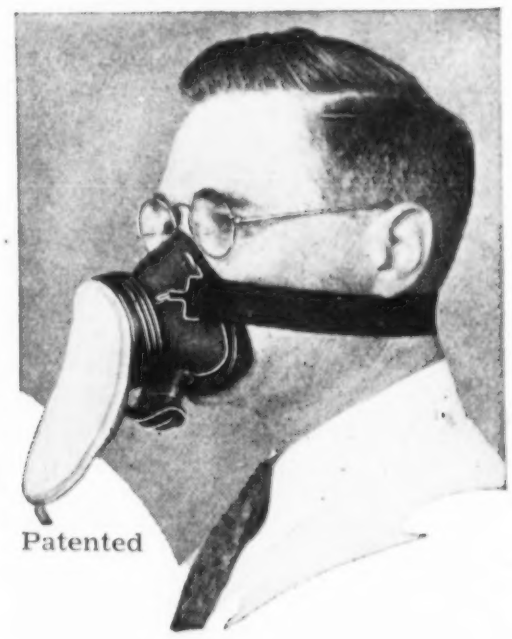
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with their schedules and special provisions.

As we all know, the first laws to stand the test of constitutionality in our country were those enacted in 1911.

Insurance companies and employers from the very beginning of this new era, and upon the passage of the first workmen's compensation laws, cooperated wholeheartedly, and are now cooperating in the development of workmen's compensation legislation. In the light of all experience available, it was soon recognized in the beginning, and still is recognized, that the very best means should be taken to make the most thorough and accurate study possible of all the problems involved in this general plan of compensation. Every conscientious effort has been and is being made to emphasize its merits and to cure its weaknesses both as to accidents and occupational diseases so that, a sound, reasonable and workable compensation system be perfected in our country.

In 1917 in compiling a report as to the general situation in our country regarding compensation legislation, I quoted from Von Jhering's "Struggle for Law" a strong statement of his to the effect that the abolition of slavery, of serfdom, the freedom of landed property, of industry, of conscience, had all been won by the most violent struggles which had lasted for centuries, and that not infrequently streams of blood and rights trampled under foot marked the way which the law had traveled—for the law was a Saturn devouring his own children.

In that report I also made the statement that there was no doubt but that another very important feature of insurance was very close upon us, which I thought would ultimately be woven into the woof and warp of our industrial development, and that was compensation for diseases. I indicated that in our country within the space of six or seven years one of the oldest branches of the law had been uprooted and disregarded in practically two-thirds of the United States by the substitution of workmen's compensation laws for the old common law of master and servant which gave rise naturally to some confusion, discontent and resistance. But I felt confident that the worker, individually and collectively, through labor organizations and physicians, attorneys, courts, boards, commissions, employers and insurance companies, who were all affected, would unquestionably meet on some common ground and by the establishment of standards of justice, each rendering to the other his just due, would rise from the then present state of bewilderment to prove the truth of Von Jhering's statement as its exception.

Parenthetically, I may say in passing, even now after eighteen years of more experience in the development of compensation legislation, I still adhere to my opinion as expressed in 1917.

SO WHEN nearly a quarter of a century ago, the first compensation laws in our country were passed, the basic principles underlying their passage and the enactment of subsequent similar laws are well known. These basic principles were then, and no doubt still are, considered sound and logical.

The reasons in which these basic principles were conceived and subsequently gave birth to this new system of compensating employees for accidental injuries, arising out of and in the course of employment, are also familiar to us all. Both the principles and reasons have been practically universally accepted, no matter whether the motive was one or all, of an economic, a humanitarian, or a utilitarian expediency.

Recently, by reason of the rapid growth and development of our complex industrial life, with its attendant acute hazards, more pronounced than ever before, intricate machinery, power-driven tools and appliances, and intensified chemical processes, we are now faced with just as serious and acute conditions in the dealings with occupational diseases contracted in employment, as we were when common law remedies for injury by accident were considered unsound, too expensive for industry and unfair to the worker.

The acute conditions we are facing today are increasing in gravity all the time. They are uncertain and extremely complicated. The greatest contributing factor to this unwholesome condition has unquestionably been precipitated by diseases caused by breathing inorganic dust, particularly the diseases of silicosis and asbestosis. These are not new diseases, but they have been alarmingly on the increase because of exposure to and the use of modern machinery and processes in everyday course of work.

I shall not attempt to minutely describe the various details outlined to us by the engineering and medical authorities as to the ramifications of many of the occupational diseases, and especially silicosis and asbestosis which are by far the most dangerous of all, nor as to the latter to dwell upon the size of the particles of inorganic dust which causes the damage; the number of those particles which can be safely breathed in a given amount of air; dust counts and methods for counting; the percentage content of silica; the stages, if they may be so called, in the development of silicosis and asbestosis; the period of time in which inhalation of this inorganic dust produces a fibrotic condition of the lungs characteristic of silicosis or asbestosis. Suffice it to say that we must all recognize this fact: That the fibrotic condition of the lungs known as silicosis and asbestosis does not develop suddenly. It is not immediately disabling as in the case when an accident occurs. These diseases creep on as thieves in the night, slowly and stealthily. Once the disease manifests itself development is sure and certain over a long period of time. In the light of research thus far made, there is grave doubt that it can be arrested and certainly thus far there is no known cure for silicosis.

CONSIDER the chaos brought about by court decisions. Just a few of these may be mentioned to prove the confliction and confusion facing us:

Occupational diseases not covered under the usual workmen's compensation policies: Policies cover accidents only:

(a) Belleville Enameling and Stamping Co. vs. U. S. Casualty Company—266 Ill. App. 586 (Ill.).
(b) U. S. Radium Corporation vs. Globe Indemnity Co. et al.—178 Atl. 271 (N. J.).

Occupational diseases sometimes held to be accidents: Negligence considered under workmen's compensation laws in determing whether or not a disability was caused by accident or disease:

(a) Victory Sparkler and Specialty Company vs. Francks—128 Atl. 635 (Md.).
(b) Gunter vs. Sharp and Dohme—151 Atl. 134 (Md.).
(c) Cambridge Mfg. Co., vs. Johnson—153 Atl. 283 (Md.).
(d) Sinsko vs. Weiskittel & Sons—163 Atl. 851 (Md.).
(e) McNeely vs. Carolina Asbestos Co.—174 S. E. 509 (N. C.).
(f) Cannella vs. Gulf Refining Co.—154 So. 406 (La.).
(g) Simmons vs. Etowah Monument Co.—157 S. E. 260 (Ga.).
(h) Tex. Employers' Insurance Assn. vs. Barron—21 S. W. (2nd) 78 (Texas).
(i) Turner vs. Va. Fireworks Co.—141 S. E. 142 (Va.).

Construction of statutes relating to occupational diseases:

(a) Burns vs. Ind. Commission—191 N. E. 225 (Ill.).
(b) North End Foundry Co. vs. Ind. Comm.—258 N. W. 439 (Wisc.).

Liability of successive employers and carriers:

(a) Blanchard vs. Ind. Comm. 228 Pac. 358 (Cal.).
(b) Plecity vs. McLaughlin Hat Co.—164 Atl. 707 (Conn.).
(c) DeFilippo's case—188 N. E. 245 (Mass.).
(d) Texileather Corp. vs. Great American Indemnity Co.—156 Atl. 840 (N. J.).

Is the compensation law the exclusive remedy?

(a) Gordon vs. Travelers Insurance Co.—287 S. W. 911 (Texas).
(b) Webb vs. Tubize-Chatillon Corp.—165 S. E. 775 (Ga.).
(c) Barrencotto vs. Cooker Saw Co.—194 N. E. 61 (N. Y.).
(d) Adams vs. Acme White Lead Works—184 Mich. 157 (Mich.).
(e) Jones vs. Rinehard & Dennis Co.—168 S. E. 483 (W. Va.)
(f) Mabley & Carew Co. vs. Lee—193 N. E. 745 (Ohio).

At common law, there was no liability for occupational diseases:

(a) Innes or Grant vs. G. & G. Kynoch—1919 Appeal Cases 765, 773 (England, House of Lords).
(b) Adams vs. Acme White Lead Works—182 Mich. 157 (Mich.).
(c) Gordon vs. Travelers Ins. Co.—287 S. W. 911 (Texas).
(d) Ewers vs. Buckeye Clay Pot Co.—163 N. E. 577 (Ohio).
(e) Sylvester vs. The Buda Co.—App. Court, 1st Dist., July 5, 1935 (Ill.).

Constitutionality of occupational disease statutes:

(a) Parks vs. Libbey-Owens-Ford Glass Co.—195 N. E. 616 (Ill.).
(b) Boshuizen vs. Thompson & Taylor Co.—195 N. E. 625 (Ill.).
(c) Boll vs. Condie Bray Glass & Paint Company—11 S. W. (2nd) 48 (Mo.).
(d) Vallat vs. Radium Dial Co.—196 N. E. 485 (Ill.).
(e) Navarro vs. Illinois Steel Company—196 N. E. 489 (Ill.).

Various other phases involved in oc-

cupational disease cases:

(a) Hendrickson vs. Continental Fibre Company—136 Atl. 375 (Del.).
(b) Dille vs. Plainview Coal Company—250 N. W. 607 (Iowa).
(c) Jerner vs. Imperial Furniture Co. 166 N. W. 943 (Mich.).
(d) Wolfe vs. Mallinchkrodt Chemical Works—81 S. W. (2nd) 323 (Mo.).

THEN witness the wholesale discharge of employees even though not disabled. Think of the avalanche of common law actions, spurious and real, for claims involving millions of dollars and, in some instances, resulting in bankruptcy to employers. Study the abnormal and adverse conditions confronting industry, the workers, State authorities, commissioners, and not only those directly affected, or interested, but the public generally.

It is only natural in this dilemma that a plan for compensation for occupational disease should be looked to as the solution to the present difficulties, just as such a plan was deemed to be the solution to the problems existing even centuries ago and certainly in modern times, when the redress was common law actions for accidental injuries sustained in employment. As to many occupational diseases, there has been, by court construction or amendments from time to time of our compensation laws, coverage granted for occupational diseases, but these diseases were then not nearly so acute nor were silicosis and asbestosis considered. The compensation laws, however, were originally intended as substitutes for the old common law and the old employers' liability laws which applied only to accidents. But the principle underlying compensation laws was that industry owes compensation for all injuries for which it could truly be held responsible without considering the question of negligence.

Therefore, if the basic principles underlying the passage of compensation laws to apply in cases of accidents are sound, the solution for the occupational disease problem may be found in appropriate legislation. A worker is just as much disabled as a result of his employment if he contracts an occupational disease which is peculiar to, characteristic of, and arises out of the work to be done in connection with specific exposures and directly related thereto as if he sustained a broken leg as the result of an accident arising out of and in the course of his employment.

The true rule is, and should be, that industry should compensate for those diseases only, which in individual cases can be medically traced to an origin in a "trade risk." That is to say, a risk not of ordinary life, but specific to and created by some process or occupation in industry. The British and all European authorities go a bit further holding that occupational diseases for which compensation is to be paid cannot practically be defined in general terms, but must be specified and based on prevailing medical opinion; that further, the terms and conditions governing the payment of compensation for such diseases can thus only be equitably required when those terms and conditions are varied to fit the needs according to the peculiarities of the different diseases.

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Just as all accidents to the worker are not compensable regardless of how, when, where or under what circumstances they happen, the test being, did the accident arise out of and in the course of employment, just so in occupational disease cases, compensation should not be paid if the disease cannot be medically traced to an origin in a "trade risk," so as to prevent communicable diseases — common colds—eye strain—tuberculosis—and many other diseases common to ordinary life, regardless of occupation, being swept in as occupational diseases.

A worker who falls from a ladder at his home while hanging a picture and breaks his leg is not entitled to compensation under the compensation act. Neither should a worker be entitled to compensation for a disease which is not strictly occupational, and which disease cannot be medically traced to an origin in a "trade risk."

If legislation providing a plan for compensation in occupational diseases is the solution, should we not endeavor to have the best legislation to carry out the bona fide intent and purpose?

Are we to find, however, the best solution by hastily amending our present workmen's compensation laws to include occupational diseases without any special provisions as to such diseases? I think not. There is such a wide divergence between accidents and diseases, as to cause and effect, time and place, immediate knowledge and ultimate result, that from every point of view it warrants the sound and logical belief that special provisions are needed to regulate and provide a plan for compensation for occupational diseases, and more especially as to silicosis and asbestosis.

The provisions of the law governing compensation for occupational diseases should differ radically from those governing compensation for occupational accidents and need to be clearly distinguished. This can be accomplished in two ways: First, by having the provisions relating to occupational diseases enacted as another part (to wit—2, 3, or 4 as the case may be) of the workmen's compensation law. Secondly, by adding a special chapter or a new and entirely separate article. By either method the provisions should be correlated in such a way as to avoid duplication from the administrative and other similar requirements which could apply to occupational diseases as well as to accidents.

THIS year 44 legislatures convened. More than 75 bills bearing on occupational diseases were introduced. At this writing new laws and amendments have been enacted in the following states:

New York—All-inclusive plan, effective September 1, 1935.

North Carolina—Schedule plan with special provisions for silicosis and asbestosis, effective March 22, 1935.

West Virginia—Silicosis only, effective March 8, 1935.

Nebraska—Occupational diseases peculiar to the smelting or metal refining industries, effective May 25, 1935.

By legislative enactments commissions are to be appointed to study the occupational disease problem in the following states: California, Maryland, New Hampshire, and Michigan.

North Carolina is the first state that has passed an occupational disease law which provides for a definite schedule, amending the compensation law with provisions dealing specifically with and applying to silicosis and asbestosis. This has been done by an entirely new section. It justifies the belief that it has been the result of an intelligent and careful study of the problem by all parties interested, and evidences a sincere effort to substantially relieve a most tense and quickly developed acute situation.

North Carolina in blazing the trail has contributed much constructively to the solution of the occupational disease legislative problem as a whole, as well as demonstrating a thorough study and understanding of its local problems.

Either in a special part, chapter or article for occupational diseases, as appropriate amendments to an existing compensation law, there are many provisions which must be considered:

Accidents are sudden events; can be fixed definitely as to time; are generally clear-cut as to causes and consequences. Occupational diseases, however are slowly occurring injuries. They are not sudden in their effect. They are not clear-cut as to causes and consequences; and they are extremely difficult to trace, because other influences disassociated with the alleged occupational disease may have led to disablement or death as intervening causes. Therefore, occupational diseases by reason of their marked difference from an injury by accident must of necessity be treated differently. They require special provisions for fulfilling the bona fide purpose and intent in making them compensable. Some of these special provisions are of major importance, others collateral, but just as essential.

The collateral provisions, although highly important, are so numerous that time will not permit a detailed discussion of them. I shall just mention some of them.

There should be definitions covering: disablement; disability; silicosis; abestosis; medical board, and similar terms.

Then there should also be special provisions as to: filing of disability and death claims; aggravation of other diseases or injuries—proration; relationship and dependency; employer responsible for compensation; average wages; medical benefits (especially as to silicosis and asbestosis because of no known cure and to avoid temptation to overtreat, or to experiment, or to gain knowledge from a research point of view); removal of employees from dusty occupations; compensation, when removed from occupation during period in obtaining other employment, if not obtainable with present employer; misrepresentation of the employee as to previous disability or exposure; subrogation; period of exposure (at least two years in state unless employee employed by same employer during the whole time within the limitation period of exposure provided for under the law); disablement or death within a fixed time after last injurious exposure; official approval of employee's engaging in employments exposing him to hazards of dust (if approval not obtained, compensation not payable); waivers; limited compensation; records, postmortems; awards; appeals.

Some major factors to be considered are as follows:

SCHEDULE plan rather than all-inclusive plan: Most of the compensation laws in European countries have schedules. Some of our states having occupational disease coverage adopted the schedule method. At present most of the schedules do not name silicosis and asbestosis, the two diseases which have recently precipitated most of the trouble. Schedules can be enlarged from time to time when new occupational diseases become known.

An all-inclusive plan may be construed to give rise to granting coverage for diseases which may not be occupational as, for example, pneumonia, bronchitis, asthma, kidney disease, heart disease, arthritis, sciatica, high blood pressure, cancer and various other illnesses common to the human race generally.

When an act gives blanket coverage by the use of the phrase "any and all occupational diseases," or similar language, it is vague, indefinite and uncertain. Commissions, boards and referees are required to make their own definitions and rulings without any legislative guidance. This leads to much litigation for court interpretation. There immediately arises doubts and uncertainties, which foster unnecessary and expensive litigation and place an added financial burden upon employer, employees, boards, commissions, courts, carriers and all interested parties.

Any all-inclusive plan, or even a schedule plan not carefully developed, is apt to give and result in such a blanket coverage of diseases generally as to make such an act tantamount to health, old age, and life insurance. This would certainly be far beyond not only the intention, but also beyond the ability of industry to provide for its workmen, indiscriminately, at the high level of benefits provided for under our compensation laws. It is often pointed out that in our country we have some compensation laws with "all-inclusive coverage" which have not been so broadly construed. This is not entirely accurate, especially in view of the experience in Connecticut, Wisconsin, California, Massachusetts, and the acute conditions in Illinois and Missouri. Employers and their insurers not knowing what they are liable for, the conditions of their liability, its limits, and probable cost, the results are that many risks are not able to get insurance and that the carriers are reluctant to provide protection because of the peril of ruinous liability.

I need not mention several specific examples, among others, as to the seriousness of the problem generally. In Massachusetts, the statutory law has practically covered all injuries indefinitely arising out of and in the course of employment since its enactment in 1912. It took much expensive litigation before it was held that silicosis was covered—with the resultant difficulties.

Witness also the confusion in Illinois where one section of the occupational disease law was held to be unconstitutional because of uncertainty and indefiniteness. Under this section the court pointed out that it was a matter of speculation as to

whom the law applied and the occupational diseases which were covered by it.

There has recently been handed down a decision in the case of Sylvester vs. The Buda Company in Illinois, holding that at common law there can be no recovery for occupational diseases. This case has been appealed to the Supreme Court and will be argued in the fall. If the Supreme Court of Illinois ultimately holds that silicosis is not caused by poisonous chemical minerals or other substances, then it does not come within the construction of section 2 of the Occupational Disease law in the Burns' case. An employee contracting silicosis may not recover either compensation or damages. He cannot recover damages under section 1 of the Occupational Disease act because that has been held to be unconstitutional.

When a schedule plan is adopted workers, employers and insurance carriers know definitely the specific diseases covered, and what they are entitled to and obliged for. This means a better approach to financial certainty for industry and for insurability. It avoids uncertainties, waste and disappointment for the workers because of speculative litigation when indefinite and general terms are used.

Dust diseases are in a special class. They are of exceedingly slow contraction. They are exceptionally difficult to diagnose and are often complicated with other diseases. The more serious of the dust diseases are progressive, incurable, tending inevitably, though slowly, to become worse. They call for special treatment from every point of view, underwriting, engineering, medical, claim, legal and statistical. It is imperative, therefore, that in any compensation plan for occupational diseases there should be special provisions to apply to dust diseases.

How much better to deal with compensation for injuries other than accidents by legislation which is specific and clear as to meaning stating exactly what it means and what diseases are covered, and upon what terms and conditions!

ACCRUED LIABILITY: Another serious problem to solve in any legislative plan is, when shall the act take effect, particularly as to silicosis and asbestosis, or any other disease which is contracted over a long period of time? When an accident occurs it is sudden. It can be definitely established as to time and place. If a compensation act becomes effective on July 1, no compensation is payable for an accident happening before July 1, even if the disability resulting from such accident occurs after July 1. As to occupational diseases, especially those of the progressive type and of slow contraction, by reason of the indefiniteness as to time, place and disability, the problem is so complicated it is difficult to avoid legislation which will not be retroactive in effect.

Insurance is protection against future hazards. Industry cannot afford to pay for losses which have accrued, and neither can the insurance carriers afford to give coverage for past liabilities accrued before the law became effective. It is practically actuarily impossible to determine and fix rates which will afford protection to an employer for his liability under the law for diseases contracted years before the law became effective, even if the intent is to give protection for the accrued liability, regardless of the legal aspect as to the retroactive feature. No reserves have been created to meet the extra burden. No premiums have been collected prior to the passage of the act with which to pay losses subsequent thereto. Reserves cannot be created and premiums cannot be charged or collected for a period of contraction when there was no coverage or liability.

As to dusty occupations, one approach to the solution may be a provision in the law for a waiting period of two or three years before it becomes effective as to benefits. In this way reserves can be provded for new obligations. All provisions as to preventive and administrative measures can be made effective soon after passage. Various State departments—labor, hygiene, health, etc.—will then have an opportunity during the waiting period to correct all hazardous conditions injurious to the health of the worker. This may not meet the full requirements, but it would unquestionably be better than to precipitate the coverage with no preparation.

COSTS: I think we are all agreed that any legislation must be such as will accomplish the objectives, and yet prescribe certain limitations which will make it possible for insurance to be obtained and the desired protection given. This must be accomplished in a way which will not prove too crushing a blow to industry. We know that compensation for silicosis and asbestosis will greatly increase costs. In some occupations and classifications involving silicosis it has been demonstrated that cost for compensation has amounted to about nine times the cost for compensation for accidents. If we add to that the cost of an all-inclusive plan there is grave danger that the cost will be prohibitive.

It is almost impossible to estimate rates. There are no statistics available to guide. Everyone should be awake to the gravity of the problem in initiating a plan of compensation for occupational diseases, and particularly the progressive type of gradual contraction. All should avoid the delusion that the problem can be solved by insurance, or otherwise, except at a very heavy cost.

A hypothetical case was worked out based on the benefits of a pending silicosis bill in New York which was vetoed by the Governor. A foundry —20 employees, four of whom were found to have the first stages of sil-

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icosis; two were found to be disabled (second stage); and 14 were in sound health. It was estimated that there should be a per capita charge of $500 making the group charge $10,000 as a deposit premium. For the four employees impaired, $1,625 per employee, group charge, $6,500; for the two disabled employees (second stage), per capita charge, $8,750, group charge, $17,500; for the 14 in sound health, per capita charge $154, group charge, $2,156—a total premium of $26,156. However, the premium on a similar risk which was found to have no impaired or disabled employees would be reduced from a deposit premium of $10,000 to a final premium of $3,080. It is to be noted, too, that the per capita charge would not likely be required after the first year of insurance, that is, after the accrued liability for the past exposure had been liquidated. This estimate was based on the limited benefits under the silicosis bill which applied only to silicosis and were not estimated under the present New York act with its full benefits for all occupational diseases, including silicosis, as for accidents. The above estimate was for silicosis alone. It does not take into account any increase in cost over the above estimate, where occupational diseases plus silicosis are to be added under the present provisions of the New York compensation act as amended, making it all-inclusive as to all occupational diseases.

At this writing two plans developed by the Compensation Insurance Rating Board of New York were submitted to the Insurance Department of New York. It was proposed that the employer and the insurance carrier by agreement be allowed to select either one of these plans which would be most suitable to the peculiar conditions of the risk.

The first plan provided for a fixed rate per $100 payroll. The employer was required to pay $300 in every case resulting in death or causing disability for four months or longer. Several rates from the schedule are: ore milling, $10; silica grinding, $20; foundries, $8; stone cutting or polishing, $18; asbestos goods, $12; cleaning buildings, $17; rock excavation, $9.

Plan two contemplated a rate per $100 pay roll. All silicosis losses were to be paid from a deposit on a per capita basis. As silicosis is progressive, based on the assumption that the time of progression covers a period of seven years, then at the time the law became effective six-sevenths of the liability for losses in the first year had already accrued.

Silicosis losses were to be paid from this fund which each employer had deposited. Payments the first year were to the extent of six-sevenths from the fund and one-seventh by the carrier. The payments the second year from the fund five-sevenths, by the carrier two-sevenths, and so on. The deposit fund was to be treated as a revolving fund to be replenished when impaired or exhausted. If such fund should exceed the amount of the claim charges, the surplus would be returned to the policyholder.

Both of these plans are the result of an effort to deal just as effectively as possible in the light of present experience with the accrued liability.

There were questions as to whether the deposit constituted a fund in the nature of a premium, or whether or not it was to be regarded as a trust; whether it would be subject to an expense loading; whether or not the fund would be taxable.

At a conference called by the Superintendent of Insurance, New York, to consider these two rating plans, industry pointed out that while it appreciated everything was being done to meet the situation fairly, yet if the proposed rates were approved there would be no risks to cover—that as to the per capita deposit, industry could not afford, after five years of depression, to make any such deposit and that the cost would force many industries to cease operations.

SPECIAL MEDICAL BOARDS: The act should provide for a medical board which would have authority lodged in it to determine all controverted medical questions. (In view

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"IT SHALL be the duty of the Division to investigate conditions of occupation with reference to hazards to health and to determine the degree of such hazards, to investigate and evaluate methods for the control of such hazards to assist in the preparation of rules and regulations for the preventing of occupational accidents and diseases, and, in cooperation with the department of public health or otherwise, to promote occupational health and safety education."—Sec. 11A of the law creating the Division, effective 1934.

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of recent developments, it would unquestionably be desirable to give such a board authority to determine all purely medical questions in claims for injuries resulting from accidents as well as occupational diseases.)

Needless to say such a board should be composed of the very best in the medical profession. The board should be appointed by the Governor, by and with the advice and consent of the senate. The physicians on such a board should devote their entire time to the board's work. The powers of this board as a fact-finding body on medical questions should be final and equal with the powers of the Industrial Commission or Accident Board in that respect.

Findings should be final and conclusive unless upon review and further evidence presented, a finding or conclusion was proven to be manifestly erroneous, or unreasonable, or due to fraud, undue influence, inadvertance, or mistake of law or fact. This board should have supervision of all examinations, if possible to make all examinations and to determine the nature and extent of disability.

PRE-EMPLOYMENT examinations; interim examinations and examinations when leaving employment in dusty occupations only; examinations for prevention and protection and not for general selective standards:

Any plan should provide for pre-employment examinations, interim examinations and examinations when leaving the employment. Such examinations, however, should apply only to dusty occupations.

Examinations should be made only for the purpose of protecting workers from the hazards of disease. Where workers are found to be suseptible to diseases to which they may be exposed, or manifest symptoms thereof, such workers should not engage in employment subject to that exposure. Such workers should be removed to another occupation wherever in the judgment of the medical board such action is deemed advisable.

No plan for physical examinations should be general in its scope. It should not apply to all workers regardless of exposure. This would savor of maintaining selective standards for all workers, which is not the intent. In other words, the sole purpose of all examinations, as provided for in the law under the jurisdiction of the medical board should be only for protection and prevention.

EXCLUSIVE remedy: When compensation laws were first passed it seemed to be generally accepted that the workmen's compensation would be the exclusive liability for occupational injuries; that the old damage suit would be gone forever. We need not spend much time debating whether or not the intention has been carried out. I think we will all agree that it has not. Much of the confusion arising today has been brought about by a revival or reincarnation of some of the old abuses.

Where compensation is provided for all recognized occupational diseases, it should be distinctly made the exclusive remedy for all "injuries by disease arising out of the employment." It is not equitable to hold employers liable, regardless of fault, for all known occupational diseases, and yet leave such employers subject to exploitation in common law suits

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for damages for other diseases unknown, or speculatively, by ingenious and far-fetched theories attributed to the employment, with the resultant needless and expensive litigation. Furthermore, the legislature, in the light of prevailing medical authorities, should determine specifically what diseases industry should be liable for, and not leave that determination to be arrived at by emotional litigation —for courts and juries are often swayed by passion and prejudice.

PREVENTION: The solution to the whole problem is to be found in prevention. The conditions brought about by the machine age are such that we must resort to the machine to cure the ills produced by machines. Remove the dust hazards and nearly all other hazards wane in seriousness.

There are two very important steps to be taken. One is to remove dust and other hazards from the work places. The other is to remove the worker from the exposure at the first indication of a manifestation of the disease, except perhaps in cases of elderly or highly skilled workers for whom a forced change of occupation might create more disatrous results than the exposure to the dust hazard.

There should be, by proper legislation, a strict and forceful regulation from an industrial, hygienic and sanitary standpoint. There should be special provisions in the law, under the proper department, to study occupational diseases; to develop ways and means of control and prevention, so that, such a body could recommend to the legislature for enactment such specific measures as would be adequate.

Regulations for the prevention of occupational diseases should emanate from a competent bureau of industrial hygiene. For only by the most thorough investigations on the part of medical boards, bureaus of hygiene, or State health departments, can there be evolved the proper prevnetive measures and regulations Unstinted cooperative efforts by legislatures, industry, labor, insurance carriers and the public generally, will be a most important step toward the best solution of all problems involved.

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